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CONTENTS

Editorials 1

Applications for Membership 7

Coming Veterinary Meetings .....

Papers:

Reproductive Hormone Therapy in Domestic Animals—G. H. Hart and H. H. Cole 12

A Study of the Channels of Brucella Infection in Bulls with Results of Conjunctival and Skin Exposures to Bovine and Porcine Strains—A. L. Delez 24

Fowl Leukosis—F. D. Patterson 32

Hemocytoblastosis and Its Relation to the Development of Fowl Paralysis and Fowl Leukemia—M. W. Emmel 45

Attempts to Isolate Brucella Abortus from the Blood of Dogs in Experimental Brucellosis—Carl Olson and William H. Feldman 51

The Toxicity of Broomweed (Gutierrezia Microcephala) for Sheep, Cattle and Goats—Frank P. Mathews 55

Does the Right Ovary of the Bovine Function More Frequently than the Left?—C. F. Clark 62

A Consideration of Urinary Calculi in Animals—C. E. De Camp 66

Clinical and Case Reports:

Skin Lesions in Cattle—W. M. Pendergast 74

Mummified Twins in a Cow—W. M. Pendergast 75

Mortality in Young Turkeys Associated with Trichomoniasis—A. G. Cierke and W. R. Hinshaw 76

Bovine Encephalomyelitis in Mexico—Javier Escalona and Fernando Camargo 81

Foreign Bodies in the Gastro-Intestinal Tract of the Dog—J. Gilbert Horning 83

Abstracts 86

Army Veterinary Service 90

Miscellaneous 92

American Veterinary Medical Association: Organization for 1935-1936 94

Association Meetings 99

Necrology 104

Personals 110

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THE kind and scope of veterinary service a country maintains is an outward expression of its governmental standards, of the wisdom of its statesmen, of the appraisal of its animal industry, of its true attitude on the conservation of indispensable resources, and of the quality of human kindness accorded to animals.

TO insure optimum results the veterinary service of a country is grounded upon the minutest details of veterinary medicine, outlined, administered and operated by a personnel with an eye on the future, qualified by the conventional curriculum.

LOSSES suffered from diseases of domestic animals in an agricultural country may be the limiting factor to its progress, a sign of national decline, or a cause of general depression; but, the extent of the losses sustained can be determined only through the operations of a well managed veterinary service.

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A SUGGESTION TO OUR STATE ASSOCIATIONS

Of the fifteen hundred or more veterinarians in the government service (U. S. Department of Agriculture and U. S. Army), a very few are members of any state veterinary association. A few are, but these are the exceptions. It is not difficult to find a reason for this condition. It is not because Bureau and Army veterinarians do not believe in supporting the state organizations. On the other hand, the fact that these men are subject to transfer, in some cases rather frequently and at short notice, probably is the most potent factor in deterring them from affiliating with some state association.

It would appear that some sort of a transferable state membership might be attractive to Bureau and Army veterinarians. It is believed that most every one would join the association in the state in which he is located, if he could feel that he would not be penalized to the extent of having to pay another membership fee the next time he is transferred to another state and applies for membership in the state association there. So, here is a suggestion.

All of the 47 state associations are now affiliated with the A. V. M. A. Many Bureau and Army veterinarians are now members of the national association. As such, these men actually have representation in the A. V. M. A. House of Representatives, through the state associations, although very few of them are members of these state organizations. This is a fact that is not generally appreciated. Why could each state association not accept the A. V. M. A. membership of a Bureau or Army veterinarian in lieu of the membership fee, whatever it might be, and enroll him as a member of the state association upon the payment of the annual dues only?

Here are the advantages of the proposed plan: The Bureau or Army veterinarian could hold membership in any state association, wherever he might be stationed, merely upon payment of the annual dues of that organization. The state association would gain from the additional members and the increased income from dues paid under such a plan. Lastly, membership in the A. V. M. A. would have an additional advantage to every Bureau and Army veterinarian. Here is a chance for everybody to win. The plan is recommended to all state associations for serious consideration.

POSTGRADUATE WORK FOR VETERINARIANS

In the report on veterinary student enrollment for the present year, published in the November issue of the JOURNAL, figures were given on graduate students but no specific comment was made on this phase of veterinary education in this country. It may surprise many to know that in the face of the largest enrollment of veterinary students in two decades, the number of veterinarians taking postgraduate work is the smallest on record. What is the reason for this? Various answers to the question suggest themselves.

The statement has been made frequently that the returns from any form of veterinary endeavor hardly justify a person spending his time and money in postgraduate study. However, this argument is not sound. We have heard it used by those who have opposed the lengthening of the regular veterinary curriculum. Then glance at the list of graduate students in history, literature, philosophy, music and astronomy, in almost any large university, and compare the returns from these vocations with those from veterinary medicine.

Has sufficient emphasis been placed on the desirability of graduate work for veterinarians? Has it been too easy for the man

with nothing more than his veterinary diploma to get a position on a faculty or laboratory staff? Just look over the present faculty lists of our veterinary colleges and note the large number of teachers having nothing but a degree in veterinary medicine, some obtained when the course was only two or three years.

During recent years the A. V. M. A. office has received quite a number of inquiries for veterinarians from institutions and organizations of one kind or another, and it would be no exaggeration to say that, almost without exception, in each case a man was wanted who had had some special line of training in addition to his veterinary course. As the president of one concern put it, "We do not want a veterinarian of the garden variety," when he explained the nature of the position the man was to fill. In this particular case the position did not absolutely require a veterinarian, but the man charged with the responsibility of locating a man for the position thought that a veterinarian with the proper background and training could fill the position better than a man without a veterinary degree.

Graduate work appears to be merely another phase of veterinary education in the United States that has been sadly neglected.

PRESIDENT FLYNN MAKING THE ROUNDS

President Flynn would be very glad to have someone tell him how it would be possible for one person to be in two or three widely separated places at the same time. There has been the usual deluge of invitations for the President of the A. V. M. A. to attend meetings of veterinary organizations in different parts of the country during the winter months and, as is the case practically every year, conflicting dates have complicated the problem of deciding just where to go.

Dr. Flynn started on his peregrinations in October by attending the annual meetings of the Eastern Iowa Veterinary Association, in Cedar Rapids, and the Interstate Veterinary Medical Association, in Sioux City, Iowa. The first week in November saw him in Atlanta, Georgia, attending the twentieth annual meeting of the Southern States Veterinary Medical Association.

During the early part of December, Dr. Flynn attended a number of meetings in Chicago, and the annual meeting of the Nebraska State Veterinary Medical Association in Columbus. In Chicago, Dr. Flynn attended meetings of the Executive Board of the A. V. M. A., the National Live Stock Loss Prevention

Board, the U. S. Live Stock Sanitary Association, and the National Association of B. A. I. Veterinarians.

This month will be an unusually strenuous one for Dr. Flynn. On January 6 and 7, he will attend the annual meeting of the Oklahoma Veterinary Medical Association in Oklahoma City. From there he will go to Columbus, Ohio, to participate in the program of the annual meeting of the Ohio State Veterinary Medical Association. Then he will jump back to Manhattan, Kansas, to attend the annual gathering of the Kansas Veterinary Medical Association. If he is able to make the necessary train connections, Dr. Flynn will proceed from Manhattan to Austin, Texas, to meet with the veterinarians of the Lone Star State in the Texas capital.

On January 20 and 21, there will be a joint meeting of the veterinarians of Tennessee and Arkansas, in Memphis, and Dr. Flynn has arranged to attend this meeting, which gives promise of being quite an affair. Then he will proceed to Meridian, for the purpose of attending the annual meeting of the Mississippi State Veterinary Medical Association. The last week in January will see Dr. Flynn in East Lansing, Mich., as a contributor to the program of the Michigan State College Short Course for Veterinarians.

Dr. Flynn has one date for February and this is with the veterinarians of Louisiana at Baton Rouge. The occasion will be a meeting of the Louisiana Veterinary Medical Association and the Louisiana State University Veterinary Short Course.

WHAT SHALL BE DONE?

It is amazing how many thoughtful men are becoming wary over the destiny of the veterinary profession. Too many of its flares of deterioration in recent years have not been studiously appraised nor acted upon in a healthy, open fashion.

A critical survey of the present situation shows that but a small percentage of the service ordinarily delegated to graduates of veterinary colleges has ever been developed and much of that which has been somewhat established through the instrumentality of the veterinary profession is falling into outside hands for administration and application. Briefly told, outsiders plan the programs and execute them as far as convenient, condescending to seek advice on the more technical problems from the college group or to invite them in to do the menial part of the task. In the face of the trend, the professional man who is honest with

himself is getting curious to know whether veterinary medicine is so worthless that the health of animals can be safely entrusted to the present setup.

At this period, the educational system charged with the development of an adequate veterinary service is breaking down. There is said to be no room in the colleges for many applicants seeking to join the professional group. The veterinary divisions of the educational institutions appear to have been singled out as the right places in which to curtail enrollment, by those who are seemingly unaware of the undeveloped state of affairs.

When the private schools closed several years ago, it was rational to predict that their successors would be enlarged and improved in obedience to the nation's requirement. Supposedly unhampered by the financial considerations which prevented the private colleges from raising standards faster than they did, encouraging recruitment to capacity and broadening the field of veterinary medicine to its proper bournes were among the hopeful anticipations to result from the change. Certainly, no one could have predicted that the change would soon bar young men from the profession.

To argue that there is a sufficient number of graduates in the United States is not to know how little of the total veterinary service is done by the college-trained group, not to understand how much of it remains to be developed, and not to realize how fast the clinical, investigational, educational and administrative functions are passing out of the hands and councils of the personnel constituting the veterinary profession.

"What shall be done?" is a question of considerable moment, one that will test the resourcefulness and strength of organized veterinary medicine. The recommendations of Hall in his presidential address of 1930 and the article, "The Veterinary Service of the United States," read before the A. V. M. A. at the 1935 meeting, have lead to the appointment of a Committee on Public Relations charged with the duty of submitting a definite plan for the operations of the service engaged in maintaining the health of animals. As the initial purpose is to make organized veterinary medicine a unit with but a single thought, the attainments of this committee are apt to be far-reaching. But, the progress made will rest to a considerable extent upon how well organized veterinary medicine is patronized by the waiting beneficiaries, and, needless to say, upon the degree of unity the national and state associations will be able to maintain. It certainly can not be questioned that the first duty of the veterinary profession is to establish a better veterinary service than the

existing one, with which the majority of veterinarians are by no means satisfied.

Judging from the sound doctrines which organized veterinary medicine in this country has always defended and the progress it has made during the short 73 years of its life, it is inconceivable that it would not mobilize its influence for the public good when an issue as important as the one now perturbing the majority of its membership has crystallized.

That improvements are needed in every branch of the service is self-evident to anyone who keeps in close touch with passing events, but no reform can be more fundamentally essential than opening the doors of the schools to qualified applicants for admission to our depleted ranks. As the veterinary profession of the present period is but the skeleton of an adequate disease-fighting unit, barring students from the veterinary schools is the greatest obstacle it has ever had to overcome. The importance of veterinary medicine is challenged by its custodians at the doors of the colleges.

L. A. M.

COLUMBUS MEMBERS ORGANIZE

With a view of getting started early on the plans for entertaining the American Veterinary Medical Association in Columbus next summer, a representative group of veterinarians located in the Ohio capital got together on December 15, and organized the Committee on Local Arrangements. The personnel follows:

Chairman: John W. Jackman, 124 S. Washington Ave., Columbus.
Vice-Chairman: O. V. Brumley, Ohio State University, Columbus
Secretary: R. E. Rebrassier, Ohio State University, Columbus.
Alumni Meetings: J. D. Grossman, Ohio State University, Columbus.
Banquet: D. W. Ashcraft, Ohio State University, Columbus.
Clinics: W. F. Guard, Ohio State University, Columbus.
Entertainment: O. V. Brumley, Ohio State University, Columbus.
Exhibits: E. P. Maxwell, 2025 S. High St., Columbus.
Finances: D. C. Hyde, State Department of Agriculture, Columbus.
Publicity: F. A. Zimmer, State Department of Agriculture, Columbus.
Registration: L. W. Goss, Ohio State University, Columbus.
Transportation and Parking: A. J. DeFosset, State Office Bldg., Columbus.

It is planned to hold meetings of the Committee on Local Arrangements monthly for the time being and, as the time of the convention approaches, more frequent meetings probably will be necessary. A glance at the personnel of the committee is all that is necessary to convince anyone that the plans for handling many of the local details of the convention are in good hands.

EXECUTIVE BOARD ELECTION

The special election now being held in Executive Board District 8 (Kansas, Missouri, Oklahoma, Arkansas, Texas and Louisiana), for the purpose of selecting a successor to Dr. J. C. Flynn, will come to a close January 13. Although unusual interest has been shown in this election, both during the primary as well as during the election proper, the fact remains that almost 100 members of the A. V. M. A. located in the six states comprising District 8 have failed to exercise their privilege of voting. In other words, over 20 per cent of the voters in District 8 have failed to cast their ballots. If we may judge from previous elections held in District 8, considerably less than one-half of these unvoted ballots could swing the election for any one of a majority of the candidates. If you are one of the almost 100 members who have failed to exercise their franchise, consider this as a final reminder to cast your ballot.

APPLICATIONS FOR MEMBERSHIP

We start off the year 1936 by listing eleven applications for membership, the same number listed one year ago. And 1935 turned out to be a pretty good year. The number of applications listed more than doubled the number for 1934, and the total was the greatest for any year since 1930. Let's make 1936 a banner year!

As is our semi-annual custom, we are quoting Section 1 of Article 2 of the By-laws, covering the manner of filing an application for membership.

> Application for membership shall be made upon a blank furnished by the Association, in the handwriting of the applicant, and must be endorsed by two members of the Association in good standing, one of whom must be a resident of the state, province or territory in which the applicant resides. Application must be accompanied by the membership fee of $5.00 and dues pro rata for the balance of the fiscal year current, as stated on the application blank. Application must be filed with the Secretary and be examined by him for correctness and completeness as far as available information will allow. After such approval by the Secretary, the latter will cause to be published in the official JOURNAL, as soon thereater as possible, said application with name and address of applicant, college and year of graduation, and names of vouchers. If no objections shall be filed with the Secretary, as against the applicant being admitted to membership in the Association, his name shall again be listed in the next issue of the JOURNAL, and if no objections shall have been filed within thirty days after the second publication of the name of the applicant, he shall automatically become a member and shall be so enrolled by the Secretary, and membership card issued. If any objections be filed

against any applicant, either on first or second notice, said application will be referred to the Executive Board for consideration.

FIRST LISTING

BACKUS, NEWELL D. 343 W. 2nd St., Elyria, Ohio
D. V. M., Cornell University, 1905
Vouchers: Reuben Hilty and E. P. Maxwell.

EINERSON, JOHN O. Lakota, N. Dak.
D. V. M., McKillip Veterinary College, 1917
Vouchers: T. O. Brandenburg and Lee M. Roderick.

EVANS, LEWIS W. La Porte, Ind.
D. V. M., Ohio State University, 1935
Vouchers: W. F. Guard and Leonard W. Goss.

FRANKLIN, HUGH L. Greeley, Colo.
D. V. M., Kansas City Veterinary College, 1918
Vouchers: I. E. Newsom and B. R. McCrory.

HASSON, DAVID S. 3026 Main St., Kansas City, Mo.
B. V. Sc., Ontario Veterinary College, 1934
Vouchers: J. C. Flynn and L. A. Merillat.

HYDE, DAVID C. 1700 Arlington Ave., Columbus, Ohio
D. V. M., Ohio State University, 1910
Vouchers: F. A. Zimmer and A. J. DeFosset.

JAMES, GEORGE W. 7521 Harrisburg Blvd., Houston, Texas
D. V. M., Chicago Veterinary College, 1920
Vouchers: Frank Hecker and J. Gilbert Horning.

LINK, ROGER P. Kansas State College, Manhattan, Kan.
D. V. M., Iowa State College, 1934
Vouchers: E. E. Leasure and R. R. Dykstra.

TOROIAN, MARK H. 11 E. Nelson St., Lexington, Va.
D. V. M., Colorado State College, 1934
Vouchers: W. L. Bendix and I. D. Wilson.

WISEMAN, EDWIN S. Post Office Bldg., Baltimore, Md.
D. V. M., Kansas State College, 1935
Vouchers: Maj. Frank H. Woodruff and Col. Robert J. Foster.

WISHARD, DELL, E. 1612 E. Alabama St., Houston, Texas
D. V. M., Indiana Veterinary College, 1911
Vouchers: J. Gilbert Horning and Archie Stallings.

Applications Pending

SECOND LISTING

(See December, 1935, JOURNAL)

Alfredson, Bernard V., 1123 Cleo St., Lansing, Mich.
Bellis, William C., Fall River Mills, Calif.
Clark, Chester F., 508 Charles St., East Lansing, Mich.
Erickson, Irwin, 7022 Brooklyn Ave., Seattle, Wash.
Lieberman, Lt. Leo L., Veterinary Station Hospital, Fort Ethan Allen, Vt.

The amount which should accompany an application filed this month is $10.00, which covers membership fee and dues to January 1, 1937, including subscription to the JOURNAL.

Oklahoma State Board of Veterinary Medical Examiners. State Capitol, Oklahoma City, Okla. January 8, 1936. Dr. Walter H. Martin, Secretary, 101 S. Evans St., El Reno, Okla.

COMING VETERINARY MEETINGS

Oklahoma Veterinary Medical Association. Skirvin Hotel, Oklahoma City, Okla. January 6-7, 1936. Dr. C. H. Fauks, Secretary, 1719 S. W. 15th St., Oklahoma City, Okla.

Wisconsin Veterinary Medical Association. Madison, Wis. January 6-8, 1936. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

Pennsylvania, Conference for Veterinarians at University of. School of Veterinary Medicine, University of Pennsylvania, Philadelphia, Pa. January 7-8, 1936. Dr. G. A. Dick, Dean, 39th St. and Woodland Ave., Philadelphia, Pa.

California State Veterinary Medical Association and University of California Veterinary Conference. University Farm, Davis, Calif. January 7-10, 1936. Dr. Cliff D. Carpenter, Secretary, 337 Central Ave., Los Angeles, Calif.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. January 8, 1936. Dr. R. S. MacKellar, Jr., 329 W. 12th St., New York, N. Y.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. January 8, 1936. Dr. Milton R. Fisher, Secretary, 4405 W. Pine St., Saint Louis, Mo.

Southeastern Michigan Veterinary Medical Association. Detroit, Mich. January 8, 1936. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Maine Veterinary Medical Association. Waterville, Me. January 8, 1936. Dr. R. E. Libby, Secretary, Richmond, Me.

Minnesota State Veterinary Medical Society. Nicollet Hotel, Minneapolis, Minn. January 8-9, 1936. Dr. C. P. Fitch, Secretary, University Farm, Saint Paul, Minn.

Cornell University, Annual Conference for Veterinarians at. New York State Veterinary College, Ithaca, N. Y. January 9-10, 1936. Dr. W. A. Hagan, Dean, Cornell University, Ithaca, N. Y.

Ohio State Veterinary Medical Association. Deshler-Wallick Hotel, Columbus, Ohio. January 9-10, 1936. Dr. R. E. Rebrassier, Secretary, Ohio State University, Columbus, Ohio.

Vermont Veterinary Medical Association. Montpelier Tavern, Montpelier, Vt. January 11, 1936. Dr. G. N. Welch, Secretary, 43 Union St., Northfield, Vt.

Ak-Sar-Ben Veterinary Medical Association. Elks Building, Omaha, Neb. January 13, 1936. Dr. J. N. McIlnay, Secretary, 3251 Leavenworth St., Omaha, Neb.

Intermountain Livestock Sanitary Association. Ogden, Utah. January 13-15, 1936. Dr. D. E. Madsen, Secretary, Utah Experiment Station, Logan, Utah.

Rhode Island Veterinary Medical Association. Narragansett Hotel, Providence, R. I. January 14, 1936. Dr. J. S. Barber, Secretary, 14 Washington St., Central Falls, R. I.

Chicago Veterinary Medical Association. Palmer House, Chicago, Ill. January 14, 1936. Dr. O. Norling-Christensen, Secretary, 1904 W. North Ave., Chicago, Ill.

New Jersey, Veterinary Medical Association of. Hotel Hildebrecht, Trenton, N. J. January 14-15, 1936. Dr. John G. Hardenbergh, c/o Walker-Gordon Laboratory Co., Plainsboro, N. J.

Kansas Veterinary Medical Association, Manhattan, Kan. January 14-15, 1936. Dr. Chas. W. Bower, Secretary, 1128 Kansas Ave., Topeka, Kan.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. January 15, 1936. Dr. T. G. Beard, Secretary, 3684 Beverly Blvd., Los Angeles, Calif.

Texas, State Veterinary Medical Association of. Driskell Hotel, Austin, Texas. January 15-17, 1936. Dr. Dee Pearce, Secretary, Box 335, Leonard, Texas.

Arkansas Veterinary Medical Association. (Joint meeting with the Tennessee Veterinary Medical Association.) Memphis, Tenn. January 20-21, 1936. Dr. T. M. Dick, Secretary, City Hall, Little Rock, Ark.

Tennessee Veterinary Medical Association. Memphis, Tenn. January 20-21, 1936. Dr. A. C. Topmiller, Secretary, Department of Agriculture, Nashville, Tenn.

South Carolina Association of Veterinarians. Jefferson Hotel, Columbia, S. C. January 21, 1936. Dr. G. J. Lawhon, Secretary, Hartsville, S. C.

Indiana Veterinary Medical Association. Severin Hotel, Indianapolis, Ind. January 21-23, 1936. Dr. W. B. Craig, Secretary, 1420 N. Alabama St., Indianapolis, Ind.

Iowa Veterinary Medical Association. Fort Des Moines Hotel, Des Moines, Iowa. January 21-23, 1936. Dr. C. J. Scott, Secretary, Knoxville, Iowa.

Massachusetts Veterinary Association. Hotel Westminster, Boston, Mass. January 22, 1936. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Colorado Veterinary Medical Association. Albany Hotel, Denver, Colo. January 23, 1936. Dr. B. R. McCrory, Secretary, Colorado State College, Fort Collins, Colo.

Maryland State Veterinary Medical Association. Medical and Chirurgical Building, 1215 Cathedral St., Baltimore, Md. January 23, 1936. Dr. Mark Welsh, Secretary, College Park, Md.

Mississippi State Veterinary Medical Association. Lamar Hotel, Meridian, Miss. January 23-24, 1936. Dr. E. H. Durr, Secretary, Clinton Blvd., Jackson, Miss.

Nevada State Veterinary Association. Reno, Nev. January 24, 1936. Dr. Warren B. Earl, Secretary, Box 1027, Reno, Nev.

Michigan State College Short Course for Veterinarians. Michigan State College, East Lansing, Mich. January 27-31, 1936. Dr. Ward Giltner, Dean, Michigan State College, East Lansing, Mich.

Missouri Veterinary Medical Association and Special Course for Graduate Veterinarians. University of Missouri, Columbia, Mo. January 28-30, 1936. Dr. Ashe Lockhart, Secretary, 800 Woodswether Rd., Kansas City, Mo.

Connecticut Veterinary Medical Association. Hartford, Conn. February 5, 1936. Dr. Edwin Laitinen, Secretary, 993 N. Main St., West Hartford, Conn.

Illinois State Veterinary Medical Association. Palmer House, Chicago, Ill. February 5-7, 1936. Dr. C. C. Hastings, Secretary, Williamsville, Ill.

Ontario Veterinary Association. Royal York Hotel, Toronto, Ont. February 6-7, 1936. Dr. H. M. LeGard, Secretary, 335 N. Main St., Weston, Ont.

Alabama Veterinary Medical Association and Short Course for Graduate Veterinarians. College of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, Ala. February 17-22, 1936. Dr. I. S. McAdory, Acting Dean, Alabama Polytechnic Institute, Auburn, Ala.

Louisiana Veterinary Medical Association and Louisiana State University Veterinary Short Course. Dalrymple Memorial Building, Louisiana State University, Baton Rouge, La. February 26-27, 1936. Dr. C. M. Heflin, Secretary, Box 1933, Baton Rouge, La.

REPRODUCTIVE HORMONE THERAPY IN DOMESTIC ANIMALS*

By G. H. HART *and* H. H. COLE
College of Agriculture, University of California
Davis, California

It has now become well established that the physiologic processes of reproduction in mammals are, for the most part, under hormone control. A large amount of knowledge has been developed within recent years regarding the origin, action and interrelation of these controlling substances. This is being added to with such kaleidoscopic activity at the present time that a special vocabulary with several synonyms for the same substance must be mastered to understand the literature appearing on the subject.

It is of great interest that internal secretory gland tissue so far removed from the genital tract as the anterior hypophysis should secrete a substance which exercises such marked effect on the gonads. It stimulates in them morphological changes and physiological activity, a part of which activity consists in secreting other hormones to carry on definite changes in the male and female accessory reproductive organs.

Evans and Bishop[1] demonstrated that the maintenance of ovulation rhythms and the capacity to have young constitute a more exacting test of a sound physiology for certain nutritive deficiencies than does growth curve alone. It is readily observed by persons experienced with any species that dysfunction of the reproductive processes can occur from comparatively slight causes and that these conditions are very widespread. Regularity of reproductive functions is of the greatest importance in our food-producing animals. The economic significance of fecundity and percentage lamb and calf crop is readily and fully recognized when cost of production studies are made on beef, cattle and sheep.

It is, therefore, not only of great scientific interest but also of real practical importance that studies of these functions and substances producing them be made in minutia. Careful attention is constantly being paid to the possibilities of harnessing the hormones for therapeutic application in correcting reproductive organ dysfunction of both sexes in man and our domestic animal species.

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*Presented at the seventy-second annual meeting of the American Veterinary Medical Association, Oklahoma City, Okla., August 27-30, 1935.

Hormones Involved

The hormones involved in reproduction are undergoing changes in their classification, new ones are being added and there are still some missing links. It is, therefore, impossible to outline the complete picture.

The most complicated list of active principles from a single gland are those from the anterior hypophysis, three of which are definitely involved in reproduction, namely, the follicle-stimulating and luteinizing substances and prolactin. The follicle-stimulating substance causes the ripening of the Graafian follicle in the female at puberty. This in turn results in the production of the female sex hormone, or estrin, which stimulates the development of the female accessory sex organs and secondary sex characters producing the changes in the uterus and vagina associated with estrum.

The rupture of the follicle in each cycle is followed by the development of the corpus luteum, which is also an organ of internal secretion producing the substance called corporin. This acts on the uterine endometrium, preparing it for implantation of the fertilized ovum and producing those changes collectively known as progestational proliferation. When pregnancy ensues, the developing placenta secretes hormones, of which estrin is most definitely established with evidence that gonad-stimulating substance also is produced or at least concentrated there in a very high degree in some species.

The development of the mammary gland in the fetus *in utero* proceeds to a certain degree probably under the stimulus of estrin. Shortly following birth, it remains relatively quiescent until puberty and pregnancy, when duct and lobule formation develop and finally, toward the time of parturition, under the stimulation of prolactin, actual milk secretion takes place.

In the male fetus, during intraüterine life, there is marked testicular development, particularly in the horse, with regression toward the end of pregnancy and following birth. As puberty approaches under the action of the gonad-stimulating substance from the hypophysis, the seminiferous tubules develop and also the interstitial or Leydig cells of the testicle and the latter secrete the male sex hormone. This in turn is responsible for the development of the secondary male sex characters as well as the accessory sex organs, notably the seminal vesicles and prostate gland.

ORIGIN, DISTRIBUTION AND PROPERTIES OF THE SEX HORMONES

Gonad-stimulating hormone: Synonyms: Gonadotropic or sex-maturing hormone, Prolan A & B, antuitrin, folluitin, follicle stimulating, luteinizing.

Smith and Engle[2] first showed the anterior hypophysis to be the origin of this substance. Smith extirpated the gland in immature rats and the reproductive organs failed to develop. Transplantation of hypophysis into immature rats on the other hand resulted in rapid development of the gonads in the animals receiving the implants. These findings were also made independently by Zondek and Aschheim,[3] in 1927. The following year, Aschheim and Zondek[4] demonstrated the presence of the sex-maturing hormone in the blood serum and urine of pregnant women. With the publication of this work, we examined the blood and urine of the horse, cow and sheep for the presence of the sex-maturing substance. It was found only in the blood serum of mares at certain stages (37th to 200th day) of pregnancy (Cole and Hart[5]).

Further work from different sources soon demonstrated that the sex-maturing hormone really consisted of two distinct substances, one being a follicle-stimulating and the other a luteinizing substance.

Fevold, Hisaw and Leonard[6] proved conclusively that the pituitary contained two gonadotropic substances and we found two substances present in mare serum.[7] Catchpole and Lyons,[8] in assaying the distribution of gonad-stimulating hormone in pregnant mare tissues, found it to be present in high concentration in the chorion and endometrium, the latter being the most potent source known when crown rump length of the fetus is 2.5 to 3 cm. This high concentration has led to the suggestion that it may be produced at this point in the chorionic epithelial cells of the placenta in addition to the pituitary gland. Significantly the endometrium of the infertile horn is relatively low in the substance, usually only one hundredth the activity of the gravid horn in the same animal. The recovery of the hormone from the endometrium requires the sacrifice of the animal at an early stage of pregnancy and the total quantity of the highly potent material in rat units is relatively small compared to that recovered from blood serum at a single bleeding.

Hypophyses of castrated animals are more potent in the substance than those from animals with gonads intact. The substance in women's pregnancy urine is not identical to that from mare serum or the anterior hypophysis itself.

Closely related substances have been found in large quantities in the urine of human beings affected with such pathological conditions as chorio-epithelioma and teratoma testis.

Estrin: Synonyms: Folliculin, female sex harmone, estrogenic substance, theelin, theelol, amniotin.

This hormone is produced primarily in the developing follicles in the ovary and brings about the changes in the female genital tract associated with estrum. These consist in enlargement of the uterus; thickening of the endometrium from cell enlargement and proliferation; marked rhythmic contraction of the uterine muscular walls; secretion of clear, unctuous mucus from the cervix, and characteristic cell changes in the stratified epithelial cells in the vaginal mucous membrane, with enlargement and congestion of the vulva.

In the last half of pregnancy it is present in large quantities in the urine of pregnant mares and other species as first shown by Zondek.[9] It is present also in the fetal gonads of the horse during their maximum growth in intraüterine life, at which time the development of pure gonadal interstitial tissue is probably larger than is to be found at any time in any other species (Cole, Hart, Lyons and Catchpole[10]).

The presence of estrin in such quantities late in gestation in the urine of mares when the ovaries are quiescent (Cole, Howell and Hart[11]) led to some speculation as to sources of production other than the Graafian follicle. Catchpole and Cole[12] demonstrated its rapid disappearance from the urine of both mare and foal immediately after parturition, suggesting a placental origin.

By ovariectomizing a mare at about the 200th day of pregnancy without disturbing the continuity of the gravid condition, estrin continued to be present in normal amounts to the end of pregnancy (Hart and Cole[13]). This, with other work by ourselves and others, pointed most forcibly to the fetal placenta as the origin of large production of estrin during late pregnancy.

It is present in very large quantities in the urine of stallions, which was confusing until studies of its chemical nature showed it to be very closely allied in molecular structure with the male sex hormone. Also the idea of antagonistic action between male and female sex hormones suggested by the work of Lillie[14,15] on the free-martin does not seem to exist.

It has been found in plants, and recent evidence shows the possibility of synthesizing the substance from plant sterols. Soybeans have been suggested as a good plant source from which to carry on synthetic production.

Corporin: Synonyms: Progestin, corpus luteum hormone.

This is the substance secreted from the corpus luteum and no other source of the hormone has been found in quantity sufficient to justify attempts at extraction or concentration. It has, therefore, been available only in the relatively small quantities obtained by chemical treatment of lutein tissue after the method of Fevold, Hisaw and Leonard.[16] Evidence points to possible synthesis from sterols of plant sources, as has already been mentioned for estrin, with which it is very closely associated in the chemical structure of its molecule.

Its function is to inhibit the effect of estrin, retard follicle development in the ovary and prepare the uterine mucous membrane for implantation of the fertilized ovum.

The male sex hormone: This substance is closely related chemically to estrin and corporin and can be synthesized from sterols of plant sources. In the animal it is produced by the interstitial cells of the testicle known as Leydig cells. Quite similar to that which occurs in the female, the gonad-stimulating substance from the anterior hypophysis stimulates the Leydig cells which produce male sex hormone. This causes the development of the secondary male sex characters and acts on the accessory sex organs, causing enlargement of the seminal vesicles and prostate gland, which results in manifestation of sexual libido.

The scrotum acts as a thermo-regulatory mechanism for the testicles and maintains these organs at a temperature lower than that of the abdominal cavity. This is necessary for the proper functioning of the seminiferous tubules in the production of spermatozoa. If the testicles are artificially insulated or naturally insulated in sheep by a heavy fleece and high condition, lowered fertility or sterility may result (Phillips and McKenzie[17]). In natural or experimentally induced cryptorchidism in mammals, the temperature of the abdominal cavity destroys the function of the seminiferous tubules. The Leydig cells are not affected, however, and thus male sex hormone is produced with resulting sexual libido. This explains why a unilateral cryptorchid stallion, after the descended testicle has been removed, still shows normal breeding ability but is always sterile.

The male sex hormone has not been available in quantity. It is interesting, as previously noted, that the urine of the stallion and the testicles contain large quantities of the female sex hormone.

Prolactin: This hormone is produced by the anterior hypophysis and is directly responsible for the actual stimulation of milk secretion. Actual secretion is the final stage in mam-

mary development which, however, has several preliminary stages in the development of its complete mechanism. As far back as 1906, Lane-Claypon and Starling[18] postulated that mammary gland development is stimulated by chemical agents and this has now been definitely proved by many workers.

Estrin and corporin have been experimentally shown in the rabbit, by Turner and Frank,[19] to both be necessary for development of the mammary gland to the point where prolactin asserts its physiological function.

The estrogenic hormone injected alone resulted in duct formation. Corporin alone had no effect in stimulating the growth of either ducts or lobules. When both were injected simultaneously, however, for 15 days the mammary gland resembled in every respect that of the normal female during the first 15 days of pregnancy. The dose of corporin necessary to produce the effect they found to be in the neighborhood of five times the amount required to produce progestational proliferation in the uterus. They then produced the normal lactation phases of the development in the second half of pregnancy by injecting extract from sheep hypophysis.

Therapeutic Application

On account of the rapid recent development of knowledge on this subject and the interrelationships of one gland to others, practically all critical work to date has been confined to experiment animals in research laboratories.

The complexity of the hormone actions and species variations are going to make it necessary for certain phases of the work to be carried out by clinicians with a knowledge of abnormalities of the genital tract and an understanding of the physiology of the reproductive processes and their endocrine relationships. The use of estrin may serve as an example in point. Although this hormone in sufficient dosage will bring about the external manifestations of estrum, it is without effect upon the ovary and does not cause ovulation to take place. Clinically, therefore, it will produce the same effect as expressing the corpus luteum, or injecting gonadotropic hormone, but these latter procedures go further and result in a new definitive follicle developing, estrus and ovulation. Likewise, the method of administration and the dosage are important. Despite desire, particularly in human medicine, for products to be orally administered, most of the hormones are ineffective when so given, because they are rendered biologically inactive in the gastro-intestinal tract. In order that worthwhile field data may be accumulated, it will be necessary for individual products containing fixed quantities of

the active substance to be administered under various clinical conditions. At the present time confusion arises from biological supply-houses placing their individual trade names on the same product which may differ as much as ten-fold in potency. This matter is receiving cognizance from the Council on Pharmacy and Chemistry of the American Medical Association and a definite name will finally be adopted for each active substance and then all should accept supplies under these names only, for uniformity and efficiency of results.

With the above limitations in mind, we will now present data on the application of the hormones to problems of reproduction in domestic animals.

GONADOTROPIC HORMONES

These substances may be obtained in quantity from the anterior hypophysis of many species, from the blood serum of pregnant mares at certain stages of pregnancy and from the urine of pregnant women. They all act on the gonads of both sexes, but the actions of these products obtained from the different sources mentioned are not identical. Those most experienced with them now believe that the difference in action depends on the relative amount of two separate hormones, one being the follicle-stimulating substance and the other a luteinizing principle. Present knowledge would lead to the conclusion that the follicle-stimulating principle works upon the cells of the seminiferous tubules and the granulosa cells of the ovarian follicle. The other is primarily concerned with the cells producing lutein tissue in the ovary and the Leydig cells in the testicle. The effect of the latter substance cannot be manifested until the follicle and seminiferous tubule cells have been stimulated to action by the former. This functional sequence and dual action has greatly retarded the accumulation of facts on these substances from the different sources. One or more of them may come into general use depending on availability, stability, physiological action, economy of production, and so on.

Our own experience has been limited to the use of active pregnant mare serum. It is, of course, desirable that studies be intensively pursued to obtain the substances in purified powdered form but mare serum containing the active principle may be used on domestic animals without serious danger from anaphylaxis in animals other than the horse, or from loss of potency in storage. We have, of course, been interested in maximal and minimal reactions with the substance when used on animals experimentally. The practitioner is interested in physiological response

as it occurs under normal conditions. The product in pregnant mare serum can be readily standardized to a concentration of from 50 to 100 rat units per cc and is apparently very constant in the relative amounts of the two substances.

In our laboratories, more experience has been obtained with sheep than any other species of farm animals.[20, 21]

The injection of this substance in doses of 100 to 500 rat units during anestrum in ewes results in gonad stimulation with follicle maturation and ovulation, from one to three follicles after three to four days, but without manifestation of heat. Evidence exists that this is the normal condition in this species during change from anestrum to regularly recurring cycles in the breeding season. A second injection, given about 17 days after the first, produces both ovulation and estrum. In some groups two-thirds or more have given this response while in others the percentage has been much smaller. We cannot state definitely whether this variation is due to differences in the physiological state of the reproductive organs in the individual animals or to a variation in the active substances injected from different batches of mare serum even though the tests on rats showed them to be the same. There is evidence from many sources that such processes as lactation and the general state of nutrition do influence the functioning of the reproductive organs. Even though the animals do not all come into estrum after two injections, the rhythm of ovarian function frequently becomes established and normal heat will occur at the end of the next 17-day period, whether or not the animals are again injected. Pregnancy following breeding in these cases has occurred in a high percentage of cases.

The procedure is valuable in this species when early lambing is desired, or a uniform lamb crop and a short breeding season would justify the time and expense. Artificial insemination, as so successfully reported by the Russians, may have a place where too many ewes are brought into heat at one time to be served by a limited number of available bucks. In the impotent ram, as in the male of other species, double the dose mentioned for the female will produce sexual libido and normal breeding, resulting in pregnancy, provided sperm are present in the semen.

The substance will not have the effect of developing spermatogenesis in males lacking normal sperm in their seminal fluid. It stimulates the germinal cells in the seminiferous tubules but these do not proceed to the development of normal sperm. The stimulus from its use acts through the increased activity of the

Leydig cells, resulting in enlargement of the seminal vesicles and prostate gland.

It is not uncommon for rams to be sexually inactive at the beginning of the breeding season and we have found a single injection of the hormone to accomplish the normal restoration of this function.

We have used this material also on the stallion, bull and boar that were impotent, and increased sexual libido resulted in a very satisfactory percentage of properly selected cases. Breeding of such treated males to females of their respective species resulted in a normal percentage of pregnancies provided, of course, that sperm were present in the seminal fluid. The dose to be injected has been empirically determined and varies, we believe, with the weight of the gonads rather than with body weight. Rats are used to assay the potency of mare serum after the method of Cole, Guilbert and Goss.[22] One rat unit is the amount that will produce from three to ten mature follicles or corpora in each of six immature female rats autopsied five days after the injection and half of which amount will fail to produce a vaginal smear of estrum in a second group of six rats.

In the cow we have given 750 rat units in a number of cases and the type of reaction produced in the ovaries of those that were autopsied for examination showed that this was about the amount required to produce a physiological reaction. The same dose has been used in the mare.

In ewes and sows, 100 to 250 rat units are considered sufficient. In the male, approximately double the dose for females is recommended. Large doses may be given in the male without untoward results but excessively large doses repeated may cause degenerative changes in the seminiferous tubules.

Overdosing in the female produces too much ovarian activity. A dose of 750 rat units in one immature heifer produced estrum in three days and on postmorten examination four rupture points were found. In six non-breeding mature cows, doses of 5,000 to 12,000 rat units resulted in excessive ovarian stimulation and ovaries as large as an orange with many follicles and corpora. Under such conditions luteinization may take place too rapidly before normal follicle rupture occurs. This is followed by the production of corporin, which is inhibiting to the action of the gonad-stimulating hormone. In the bitch, having only two periods of estrum a year, the persistence of the activity of the corpora will probably prevent an injection of the gonad-stimulating hormone from manifesting its action for some time after

the beginning of metestrum. We do not have any data on dogs covering this point.

In wild animals in captivity, we have one personal communication regarding its use on a fox farm. In this species males often fail to serve rutting females. In some five or six cases of this kind, unresponsive males served their mates as early as 24 hours after the injection of the hormone.

In nutrition studies in our laboratories, male rats were kept on a normal diet until 60 days of age and then were transferred to a low protein diet. These animals usually failed to breed females after being on the deficient diet for two to three months, during which time there was a significant loss in weight. Epididymides of these animals still showed motile spermatozoa. Gonadotropic hormone in mare serum was injected into nine of these males, each of which had been previously tested from four to twelve times on females in heat, with only one positive mating obtained. The dose of serum was probably excessively high, being 5 cc, containing 50 to 100 rat units per cc. Positive matings were obtained on the third or fourth day with the exception of one male. As many as seven positive matings were secured from individuals tested over a period of 15 days. In all, 30 positive matings were obtained from the group, 15 of which were fertile and 14 of these resulted in the birth of normal young (Cole, Guilbert and Goss).[22] On postmortem examination, the seminal vesicles and prostate gland of injected rats are always found to be increased in size over littermate controls.

From the above observations and data and with judgment in selection of cases the gonad-stimulating substance is indicated in cases of impotency in males of any species of Mammalia, as well as in females that fail to come in heat.

Estrin: This substance is now available on the market, but its cost has been too high for general use in the treatment of domestic animals. Where it has been used in animals as well as in human medicine, the dosage has been too low in many cases for results to be expected. This hormone in sufficient dosage stimulates the uterine musculature to most marked rhythmic contractions. For this reason it may have a place in causing the evacuation of the uterine contents in cases of pyometra and in increasing uterine muscular tone. Its other effect of increasing epithelial cell activity in the uterine, cervical and vaginal mucous membranes would tend to overcome inflammatory conditions. It has no known direct effect on the ovary and it is highly improbable that repeated injections will start the abnormal genital tract to its normal rhythmic physiological activity. It is being used

more and more widely in human medicine as clinicians become better acquainted with its action.

Corporin: Corporin and male sex hormone are available only in limited quantities and little clinical data are available regarding their indications and responses.

Prolactin: This is a highly potent hormone available in concentrated form and its potency can be tested on the crop glands of pigeons. Its function is to stimulate actual milk secretion after duct and lobule formation have taken place. It would appear to have a place in bitches or females of other species that fail to produce milk following parturition. In dairying, to increase the normal milk-flow of a lactating cow, it probably has no place. Little clinical data are available.

In conclusion, all of the known hormones of reproduction are just passing from controlled laboratory studies to field use. The latter is still distinctly in the experimental stage. Care is needed in selecting the product to be used, as well as the dose and the time intervals between repeated doses. When these are properly considered and cases are carefully selected, interesting and valuable results can be expected from the use of these products as therapeutic agents.

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Discussion

DR. H. E. KINGMAN: It must be apparent to most of us that the veterinary profession is gradually emerging from the stage in which guess-work can be depended upon. It has been very difficult for the veterinary profession to correlate the physiologic and the pathologic findings with practice. I think most of us have dismissed our fundamental training in regard to physiology, histology and pathology and

have depended upon too much guess-work and mere clinical results without an effort to base our therapy and our handling of cases upon fundamental principles. Today we are faced with the necessity of understanding the fundamental principles relative to reproduction, or else we will be unable intelligently to handle and use the therapeutic agents that will come from such work as Dr. Hart and Dr. Cole have done.

The Outlook for Hogs

Although supplies of hogs for slaughter are expected to decrease during the next few months, by April supplies will begin to increase until the end of the marketing season in the fall, unless the forecasts of the U. S. Department of Agriculture are all wrong. Further improvement in the domestic consumer demand is expected, but there is little hope for any improvement in foreign export business. Hog prices until next September are expected to average higher than they did in 1934-1935, but they will not reach the peak prices of the past summer, it is believed.

During the fiscal year 1934-1935, the number of hogs slaughtered under federal supervision was smaller than for any year in the last quarter of a century. It is predicted that the number of hogs slaughtered during the fiscal year 1935-1936 will be even smaller.

Imported Cattle Must Be Free from Bang's Disease

Cattle imported into the United States from countries other than Mexico must be tested and found negative for Bang's disease, unless they are brought in for immediate slaughter, according to a new regulation of the U. S. Department of Agriculture, which became effective January 1, 1936. Mexico is not included under the new regulation because the United States has a treaty with that country relating to the health of imported animals. The new regulation, designated as Amendment 1 to B. A. I. Order 352, applies to all cattle six months of age, or older, that are imported for either breeding or milking purposes.

Cattle tested in a foreign country and found negative for Bang's disease may be imported if they are accompanied by the proper certificates. Another provision is that cattle may be tested by an inspector of the U. S. Bureau of Animal Industry at the port of entry into this country, if the animals have not been tested for Bang's disease officially. Any animals that react to the test at the port of entry will not be admitted, but must be returned by the shipper or slaughtered.

A STUDY OF THE CHANNELS OF BRUCELLA INFECTION IN BULLS WITH RESULTS OF CONJUNCTIVAL AND SKIN EXPOSURES TO BOVINE AND PORCINE STRAINS*

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Considerable attention has been given to the modes of transmission of the bovine type of Brucella in the bull. Lubbehusen and Fitch[1] reported the production of high agglutinin titres following repeated exposures by the mouth and repeated intravenous injections. Seddon[2] recovered the Bang organism from the testicle of a bull which received an intravenous inoculation when three months old. The bull was killed six months following the exposure. M'Fadyean, Sheather and Minett[3] obtained a 1:200 agglutination reaction in one bull by injecting the organism into the prepuce.

Investigations have shown that other portals of entry, namely, the conjunctiva and the skin, either intact or abraded, may play a part in the transmission of brucellosis. Schroeder and Cotton[4] infected guinea pigs with the bovine Brucella strain by intraocular instillations. Cotton and Buck[5] have reported successful attempts to produce Brucella infection in susceptible cattle by conjunctival exposure.

The infection of guinea pigs was demonstrated by Hardy, Hudson and Jordan[6] and Everson, Poelma, Brueckner and Pickens,[7] following exposure to bovine and porcine strains either through the intact or abraded skin. More recently, Cotton, Buck and Smith[8] have succeeded in transmitting Bang's disease to pregnant susceptible cattle both through the intact and abraded surface of the skin when using bovine types of Brucella.

A study of the possible transmission of Brucella infection in bulls by conjunctival and skin exposures was deemed of sufficient importance to merit attention.

In the present experimental work three young bulls were given intravenous injections of Brucella strains. Each bull received a 48- or 72-hour growth of an ordinary agar slant suspended in 5 to 10 cc of physiological salt solution. Bull 1 was injected at four months of age with a bovine strain which was two years old. Bull 2 received a similar exposure when three months old, using a strain isolated from the milk of a cow six years pre-

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viously. This strain had been used to expose cattle experimentally because of its constant pathogenic properties. Bull 3 was exposed when six months of age to a porcine strain isolated three years previously and which showed a considerable degree of pathogenicity for guinea pigs.

Bulls negative to the agglutination test and which had attained the age of puberty or mature bulls were used for conjunctival and skin exposures. It was also attempted to select more recently isolated strains for this purpose.

Conjunctival exposures usually were made by introducing into the inner surface of the lower eyelid from one to four drops of a dense suspension of organisms washed from a 48- or 72-hour agar slant culture. Bulls 5 and 7 were exposed by the conjunctival method to bovine strains which were one year and eight months old, respectively. Bull 12 was exposed to a bovine strain which had just been isolated from the placenta of a cow which had aborted from natural exposure. Bulls 4 and 10 received conjunctival exposures to porcine strains recently obtained from swine fetuses of field cases of abortion.

Skin exposures, usually at intervals of two weeks, were made on the surface of the scrotum and on the skin of the lumbar region. In the case of the latter site, a small area was clipped carefully but not abraded. Applications were made by placing cotton pledgets containing the moist growth from a 48-hour culture of a large agar slant. Several thicknesses of muslin were placed over the cotton pledget and this in turn was held in position with wide adhesive tape. The bulls were kept in stanchions, on clean, concrete floors, for 18 hours following each exposure, when the applications were removed and the animals were allowed to run in a lot in which there were no other cattle. Bulls 9 and 11 received exposures to a bovine strain which had been used previously for a conjunctival exposure for bull 7. Bull 8 received three exposures on the intact skin of the back at intervals of eleven days between the first and second exposures and eight days between the second and third exposures.

Thirteen days following the third exposure, the organisms were deposited on the abraded skin of the scrotum. Bull 11 received the same series of exposures as bull 9 except that all were applied on the scrotal surface. Bulls 8 and 10 were exposed to a recently isolated porcine strain. Bull 8 received seven exposures on the scrotal skin. The first three exposures were on the intact skin with an interval of eleven days between the second and third applications. Thirteen days later, a series of four exposures on the abraded scrotum were given at 16-, 5- and 8-day

intervals. Bull 10 was given a series of six exposures on the intact skin of the back at intervals of 13, 8, 13, 16 and 3 days. Eight days later, an exposure was made on the abraded scrotum, followed by a similar exposure at an interval of 26 days. This bull was kept for two months, when two conjunctival exposures were given 22 days apart, using the same strain as in the case of the skin exposures.

In order to observe the effect of feeding the porcine strain, bull 6 was drenched with 300 cc of physiological salt solution containing a 48-hour agar culture from a 32-ounce flask.

Results

Table I gives a summary of the serological, bacteriological and histological data, as well as the methods of exposure, strains used, and the ages of the bulls when exposed and killed.

Intravenous exposures: The serum of bull 1 reacted in a 1:100 dilution two months following exposure. Four months later, before the animal was killed, the titre was 1:50. Bull 2 had a 1:100 serum titre five months after exposure and it dropped to 1:50 one month later and finally to 1:20 between the eighth and tenth months following exposure. The titre arose to 1:100 one year following exposure. A 1:1,000 titre was observed in bull 3 two months following the exposure to a porcine strain. One month later, the titre dropped to 1:200 and the serum reacted at 1:200 and was incomplete at 1:500 four weeks before the animal was killed. No organisms were recovered by cultural or guinea-pig inoculations from the three bulls exposed intravenously. Tubular degeneration was found in the testicles of each animal. Bull 2 showed a slight focal interstitial epididymitis of the left epididymis.

Conjunctival exposures: Bull 5 gave an incomplete serum agglutination at 1:20 six weeks following intraocular instillation of two drops of a dense suspension of a bovine strain. The titre did not rise when observed for four months following exposure. Bull 7 received a similar exposure and a maximum serum titre of 1:100 was observed three months later. The animal was kept for two months and the titre remained the same. Bull 12 developed a 1:500 titre three weeks following the first exposure to a recently isolated bovine strain. The titre was the same two weeks later, when the animal was killed. Bull 4 developed a 1:50 titre ten days following conjunctival exposure to a porcine strain. The serum gave an incomplete reaction in a 1:25 dilution two months later. Bull 10 developed a 1:50 titre following a similar exposure. Cultural examinations and guinea-pig inoculations gave negative

results in all the bulls in this group. Bull 12 showed bilateral focal interstitial epididymitis and evidence of necrotic changes in the left testicle.

Skin exposures: Bulls 9 and 11 did not show serum agglutinins following repeated attempts to produce infection by placing a bovine strain on the intact skin. Bull 9 showed a serum agglutination of 1:50 two weeks after the organism was placed on the abraded scrotum. A similar exposure produced a 1:25 serum agglutination in one week in bull 11. Repeated exposures to a porcine strain on the scrotal skin, both intact and abraded, failed to yield an agglutinin response in bull 8.

Bull 10 gave a serum agglutination of 1:25 following four exposures to a porcine strain on the intact skin of the back. An incomplete agglutination in a 1:25 dilution was observed for seven weeks. Repeated skin exposures did not result in a higher serum titre. Ten weeks later, the serum was negative in a 1:25 dilution. Subsequently, as described above, this bull received two conjunctival exposures and a maximum titre of 1:50 was observed.

Bull 6, which was fed a large amount of Brucella organisms of the porcine type, did not show evidence of infection when killed 38 days later.

Discussion

Of the bulls receiving intravenous exposures, bull 2 had been nursing a dam with a colostrum having an agglutination titre of 1:200. This bull had a 1:500 serum reaction at three months of age, when the injection of the bovine strain was administered. The original titre was apparently only passive in nature, since five months later the serum gave a 1:100 agglutination reaction. When about ten months of age, this bull successfully bred several heifers which indicated the fertility was not appreciably impaired.

The highest agglutination titre in this study was observed in bull 3, which received an intravenous injection of a porcine strain. The titre dropped in one month, however, and seemed to remain stationary at 1:200.

No agglutinin response was observed in bull 5 following a conjunctival exposure to a bovine strain. This bull was eleven months old when exposed, while bulls 7 and 12, which received similar exposures, were two years and 18 months of age, respectively, when exposed. A delayed agglutinin response was observed in bull 7, the first reaction, a 1:100 titre, being observed three months following exposure. The same titre was observed

TABLE I—*Data on bulls exposed to bovine and porcine Brucella strains.*

BULL	AGE (MOS.)		METHOD OF EXPOSURE	STRAIN	AGGLUTINATION TITRE		BACTERIOLOGICAL EXAMINATION	HISTOPATHOLOGY
	EXPOSED	KILLED			BEFORE EXPOSURE	AFTER EXPOSURE		
1	4	11	Intrav.	Bovine	—20	+100	Testicles and epididymides sterile	Testicles—tubular degeneration. Epididymides—dearth of spermatozoa
2	3	17	Intrav.	Bovine	+500	+100	Testicles and seminal vesicles sterile	Testicles—tubular dengeneration. Left epididymis—focal lesions
3	6	13	Intrav.	Porcine	—20	+1000	Testicles and seminal vesicles sterile	Testicles—tubular degeneration. Epididymides—dearth of spermatozoa
4	8	14	Conjunct.	Porcine	—20	+50	Testicles—sterile. Inguinal glands—negative	No sections
5	11	18	Conjunct.	Bovine	—20	±20	Testicles and inguinal glands sterile	No changes
6	9	11	Per os	Porcine	—20	No record	Testicles and inguinal glands sterile	Left testicle—tubular degeneration. Left epididymis—dearth of spermatozoa
7	24	29	Conjunct.	Bovine	—20	+100	Testicles and seminal vesicles sterile	Testicles—tubular degeneration
8	13	15	Intact scrotum—3 Abraded scrotum—4	Porcine	±25	—25	Testicles and epididymides sterile	No changes

TABLE I—*Data on bulls exposed to bovine porcine Brucella strains—Continued.*

BULL	AGE (MOS.)		METHOD OF EXPOSURE	STRAIN	AGGLUTINATION TITRE		BACTERIOLOGICAL EXAMINATION	HISTOPATHOLOGY
	EXPOSED	KILLED			BEFORE EXPOSURE	AFTER EXPOSURE		
9	10	12	Intact lumbar—3 Abraded scrotum—4	Bovine	—25	+50	Testicles and epididymides sterile	Epididymides—tubular degeneration
10	13	19	Intact lumbar—6 Abraded scrotum—1 Conjunct.—2	Porcine	—25	+50	Testicles, epididymides and seminal vesicles sterile	Seminal vesicles—foci of lymphocytic infiltration
11	13	15	Intact scrotum—3 Abraded scrotum—1	Bovine	—25	+25	Testicles and epididymides sterile	Testicles—tubular degeneration
12	18	19	Conjunct.—2	Bovine	±25	+500	Testicles, epididymides and seminal vesicles sterile	Left testicle—necrotic focus. Epididymides—focal epididymitis

Key: +50 = Complete agglutination in 1:50 dilution. —20 = No agglutination in 1:20 dilution. ±25 = Incomplete agglutination in 1:25 dilution. Intact scrutum 3 = exposed 3 times on intact surface of scrotum.

Note: Sera of bulls 1 to 7, inclusive, tested with antigen comparable to tube 3 of McFarland nephelometer and bulls 8 to 12 with tube 1 of same scale.

two months later when the animal was killed. The epididymal lesions of bull 12 were suggestive of Brucella infection. This bull was exposed to a bovine strain which was of more recent isolation than the other bovine strains used in this experiment. Bulls 4 and 10, which were exposed to porcine strains through the conjunctiva, did not show as high agglutination reactions as the bulls similarly exposed to bovine strains with the exception of bull 5.

The skin exposures to a bovine strain showed agglutinin response only when made on the abraded surface. Bull 10 gave only a slight indication of agglutinin response to the porcine strain when placed on the intact skin.

SUMMARY

Intravenous injections of Brucella cultures in two young bulls did not result in positive evidence of infection. One bull showed a slight focal interstitial epididymitis. A six-month-old bull, which received a porcine strain intravenously, developed a 1:1,000 serum titre but no other evidence of infection was obtained.

Greater agglutinin response occurred in bulls exposed through the conjunctiva to bovine strains than to porcine strains.

Intact skin exposures resulted in practically no agglutinin response to either bovine or porcine strains. Agglutination reactions followed the deposition of bovine strains on the abraded skin of the scrotum.

No Brucella organisms were recovered from the genitalia of bulls which were exposed to bovine or porcine strains through the conjunctiva or skin. An 18-month-old bull, which received conjunctival exposure to a recently isolated bovine strain, developed a serum agglutination titre of 1:500 in three weeks. Histological examination showed a well-marked focal interstitial epididymitis.

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Hearing on Serum Marketing Agreement

The Secretary of Agriculture has announced a hearing on a proposed marketing agreement for the handlers of anti-hog cholera serum and hog cholera virus, to be held at the Fontanelle Hotel, Omaha, Neb., January 13, at 9:30 o'clock.

The proposed marketing agreement has been drafted by representatives of the serum industry in consultation with the U. S. Bureau of Animal Industry, and if it becomes effective, it will be administered by the Bureau. The agreement has been drafted under an Act of Congress, Public No. 320, approved August 24, 1935, which relates to the handling of serum and virus in interstate and foreign commerce.

Copies of the proposed agreement may be obtained from the Chief Hearing Clerk, Agricultural Adjustment Administration, Washington, D. C.

Purebred Horses Imported

There were 274 purebred horses imported into the United States during the year ended June 30, 1935, according to figures released by the U. S. Department of Agriculture. There were representatives of nine breeds among the importations:

American Saddle	1	Hackney	52
Arabian	6	Percheron	35
Belgian	89	Standardbred	3
Cleveland Bay	4	Thoroughbred	66
Clydesdale	18		
		Total	274

Bureau Transfers

Dr. C. W. Bean (Colo. '35), from Beltsville, Md., to Tallahassee, Fla., on Bang's disease eradication work.

Dr. Gordon C. Kendall (Ala. '28), from Montgomery, Ala., to Lincoln, Ill., on avian tuberculosis eradication.

Dr. R. R. Newman (K. C. V. C. '08), from Madison, Ind., to Allentown, Pa., on meat inspection.

Dr. W. C. Dye (K. C. V. C. '08), from Denver, Colo., to Kansas City, Mo., on meat inspection.

Dr. A. E. Wardlow (O. S. U. '17), from Columbus, Ohio, to Piqua, Ohio, on meat inspection.

FOWL LEUKOSIS*

By F. D. PATTERSON, *Auburn, Ala.*
College of Veterinary Medicine, Alabama Polytechnic Institute

The material which forms the basis for this paper was derived principally from our research studies conducted as a coöperative project between the Veterinary Research Institute and the Poultry Husbandry Subsection of the Iowa Agricultural Experiment Station, Iowa State College, Ames, Iowa. These data have been tabulated in two previous papers.[1, 2] One of these[1] also contains a review of the literature. For these reasons we have omitted a retabulation of our research data, and except for a few of the more important publications which have come to our attention since our last paper was completed, the literature is not reviewed in this paper.

REVIEW OF RECENT LITERATURE

Johnson[3] has presented a very interesting bulletin, "The Etiology and Histogenesis of Leukosis and Lymphomatosis of Fowls." He concluded that fowl paralysis, neurolymphomatosis gallinarum, erythro- and myeloid leukosis were all produced by a common filtrable agent. Stubbs[4] reported some valuable studies on the transmission of fowl leukosis by the use of dried blood. His experiments indicate that dried blood from fowls affected with leukosis may remain infective for at least 932 days. Stubbs and Furth[5] have reported some very interesting studies on the relationship of leukosis to sarcoma in fowls.

PATHOLOGY

The various types of fowl leukosis are by no means always clear cut; in many cases they seem to merge or blend into each other. They show many modifications and variations and numerous combinations of types are noted. However, in order to facilitate description, it seems desirable to divide them into the following types:

1. Myeloid.
2. Erythroid.
3. Lymphoid.
4. Nerve.
5. Eye.
6. Mixed.

The myeloid type is characterized by the infiltration and proliferation of myeloid tissue in the affected parts. This type can be positively determined only when a considerable number of

*Presented at the seventy-second annual meeting of the American Veterinary Medical Association, Oklahoma City, Okla., August 27-30, 1935.

the immature cells composing the pathological tissue show positive differential tendencies toward the granulocyte series. This is indicated by the appearance of granules in the invading cells, such as noted in promyelocytes, myelocytes and intermediate forms. In our most outstanding myelocytomatous cases, the neoplastic tissue was loosely constructed, soft and very light gray to practically white in color, often showing a pinkish tint. This type of tissue has been found in many different parts of

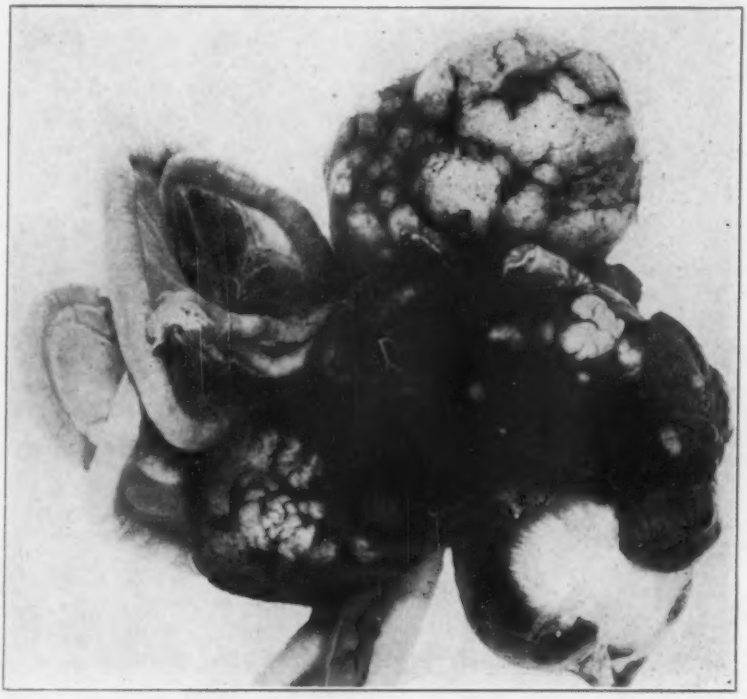

FIG. 1. Liver and spleen of lymphoid leukosis case. Note neoplastic tissue in liver and enormously enlarged spleen, which is practically replaced by neoplastic tissue. (Case S529.)

affected fowls including the liver, bone-marrow, spleen, kidneys, apex of sternum, intercostal spaces, pancreas, intestine, thyroid, parathyroid, trachea, ovary, bone, and meninges of the brain. Myeloid tissue may appear in nodular or diffuse forms.

Probably the most constant findings in typical myeloid-type cases are noted in the liver, spleen, kidneys, bone-marrow and blood. The liver, spleen and kidneys are often uniformly enlarged, swollen and of a reddish brown or grayish brown color

and diffusely infiltrated with myeloid tissue. The bone-marrow becomes much increased, more solid in consistency and is pinkish gray in color. This is due to myeloid hyperplasia. In one case the bone-marrow, from gross examination, was apparently replaced by firm, gray, lymphoid-like, neoplastic tissue, but a histologic examination indicated a myelocytomatous hyperplasia. In two other cases the bone-marrow resembled powdered chalk that had been moistened. The color was practically white with

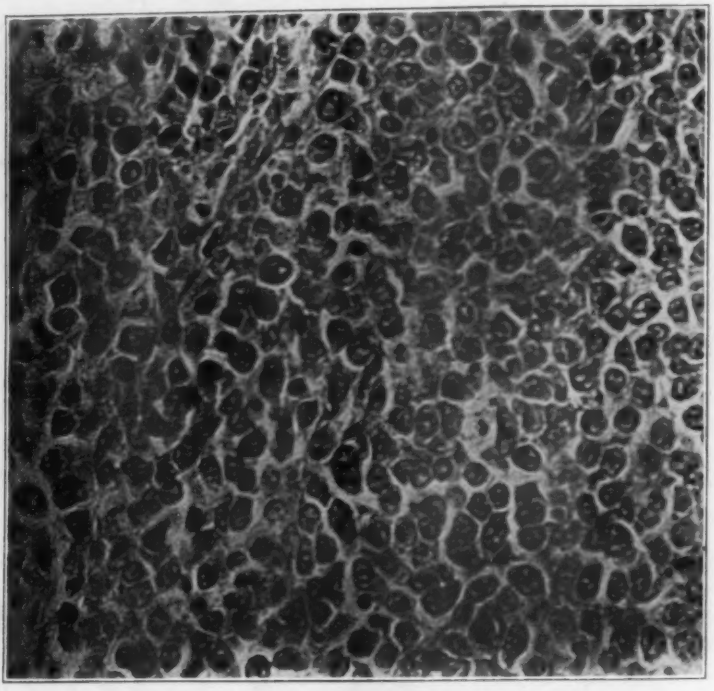

FIG. 2. Microscopic study of section from lymphoid neoplastic tissue from case S529.

but a faint pinkish suggestion. The bone-marrow in long-standing, chronic cases often shows a tendency to become ossified. In some cases the marrow cavity is almost obliterated by the formation of bone-like tissue.

The blood shows considerable variation in different cases and may do so in the same case at different stages in the course of the disease. The blood picture may be aleukemic or very markedly leukemic. All degrees between these two extremes are noted. The typical blood pictures in this type show an enormous increase in cells of the granulocyte series, a large

percentage of which are unripe forms such as myeloblasts, promyelocytes and myelocytes. Some cases show more of a qualitative involvement, while others are more of a quantitative one. Apparently normal blood pictures are noted. The red cells are frequently involved in this type, showing a tendency toward erythroleukosis, sometimes to such an extent that a clear-cut

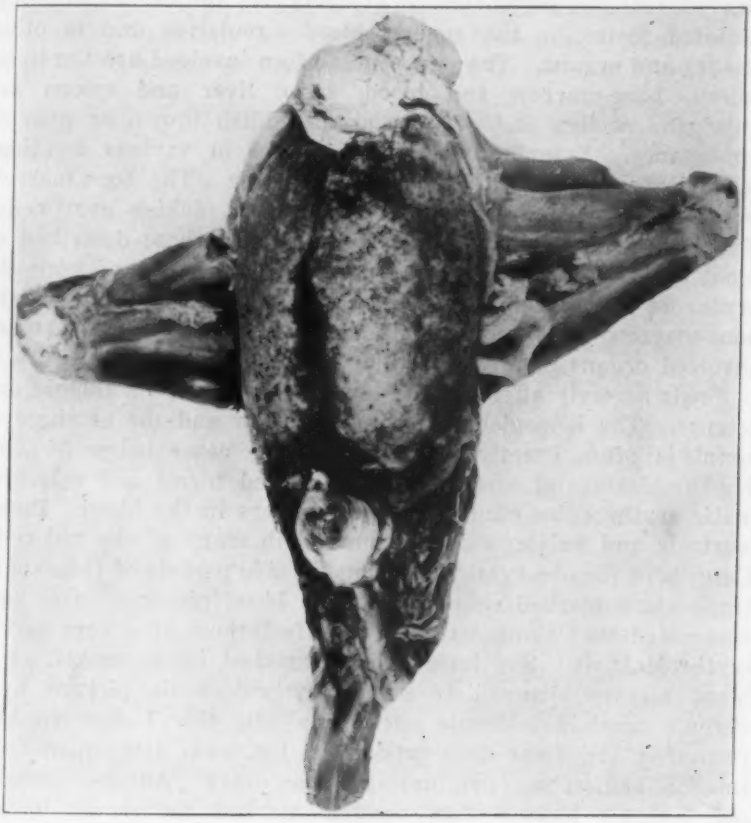

FIG. 3. Liver of lymphoid leukosis case.

separation into any one particular type is impossible. Occasionally cases are noted in which the predominant cells in the blood are large, undifferentiated, basic staining, mononuclear cells. Unless a sufficient number of other unripe cells show their differential tendencies, it is impossible to classify them except as stem cells, hemocytoblasts, which means that the undifferentiated cells may possess potential tendencies toward either myeloid, erythroid or lymphoid-type cells.

We can see no reason why the aleukemic myelocytoma cases should be separated from myeloid leukosis cases in which the blood shows a leukemic condition and have included them in the myeloid leukosis group.

The erythroid type, erythroleukosis, is characterized by the occurrence of premature unripe cells of the erythroid series, such as erythroblasts, polychromatic erythrocytes and earlier undifferentiated forms, in the general blood circulation and in other tissues and organs. The parts most often involved are the liver, spleen, bone-marrow and blood. The liver and spleen are enlarged, swollen and of a peculiar reddish brown or grayish appearance. Lymphoid neoplastic tissue in various locations often is associated with the erythroid type. The bone-marrow becomes hyperplastic, more solid and of a pinkish gray color. The bone-marrow, liver and spleen resemble those described as most typical of the myeloid type. In many cases of both the erythroid and myeloid types there is a marked tendency for the bone-marrow to simulate in color and consistency the other involved organs of any particular case.

Fowls severely affected with erythroleukosis show anemia and icterus. The hemoglobin readings are low and the erythrocyte count is often greatly reduced, in some cases below 500,000. Erythroblasts and earlier undifferentiated forms and polychromatic erythrocytes occur in large numbers in the blood. Poikilocytosis and anisocytosis are marked in many of the red cells. Cases kept for observation for considerable periods of time sometimes show marked changes in their blood pictures. One case was noted that showed typical manifestations of a very severe erythroleukosis. She later showed marked improvement. Her blood picture changed from an erythroleukosis picture to a chronic myeloid leukemic one. Clinically this hen apparently recovered and came into production but soon after died from internal hemorrhage originating at the ovary. Autopsy revealed old leukosis lesions that showed marked tendencies toward healing.

A typical erythroleukosis blood condition may change later to such an extent that the predominating pathological cells are undifferentiated forms that resemble unripe pathologic lymphocytes so closely that had it not been for the earlier studies a diagnosis of a lymphatic leukemic blood condition may have been made. Such cases may show typical lymphoid leukosis lesions when autopsied.

The lymphoid type is characterized by diffuse or nodular lymphoid neoplastic formations in the affected parts. Practically

any organ or tissue may be involved. The extent of involvement varies greatly in different cases. Some show only small foci confined to one or a few locations while others show very marked generalized involvement, the neoplastic lesions often reaching enormous proportions. Probably the most common locations are the liver, nerves, kidneys, spleen and ovary. The lesions in this type are grossly and histologically identical with the lesions variously termed lymphatic leukemia, lymphocytoma, round-cell sarcoma and lymphosarcoma.

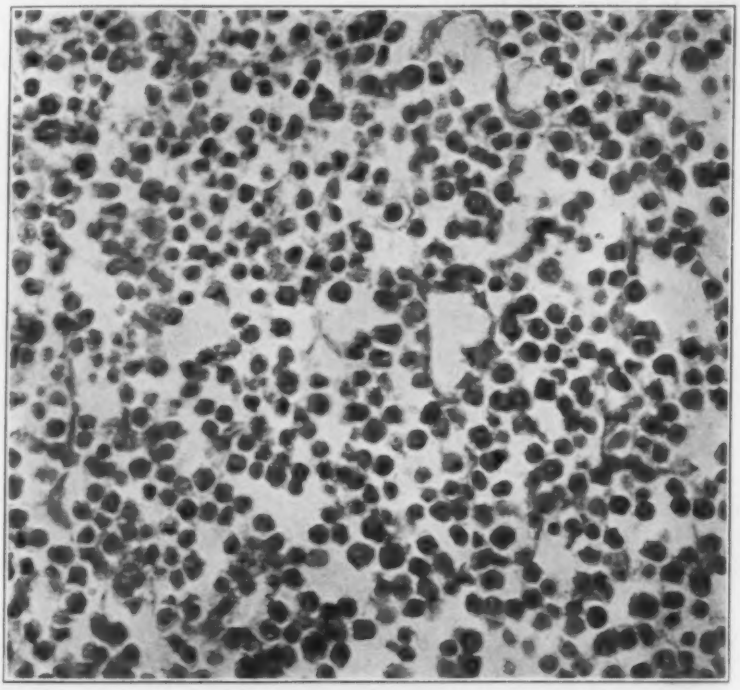

FIG. 4. Microscopic study of section from nerve affected with fowl leukosis.

The type cell that characterizes the lymphoid type is the lymphoblast. This is in general a large, round, non-granular, basic-staining cell. However, they often show marked variations in shape and size, especially in young, active, highly malignant neoplastic tissue.

The hemocytoblast is the common stem cell, progenitor of all the different cells which characterize all types of fowl leukosis. This cell is identical in appearance to the early lymphoblast, therefore, since the cells of the lymphocyte series contain

neither granules, as in the cells of the granulocyte series, or homoglobin, as in the cells of the erythrocyte series, their differential tendencies are often very obscure and a positive classification upon morphology and staining characteristics is frequently impossible.

Grossly the young, active, pathologic, lymphoid, neoplastic tissue is gray or yellowish gray in color. It is comparatively soft, though well organized. Fibrosis, in varying degrees, is often evident in the older lesions. In such cases the neoplastic tissue is firmer and harder.

The blood in the purely lymphoid type is aleukemic, though a severe lymphocytosis or an eosinophilia is often noted. This

FIG. 5. Section through brachial nerve plexus and brachial region of spinal cord of nerve-type leukosis case. (Low power.)

type is frequently combined with the erythroid type, in which case the bone-marrow and the blood findings correspond to those discussed under the erythroid type.

The bone-marrow in the purely lymphoid-type cases usually shows no significant changes, though small, multiple, lymphoid-like, neoplastic areas have been noted.

The nerve type properly belongs to the lymphoid type, as the pathologic tissue involved is identical to that of the lymphoid

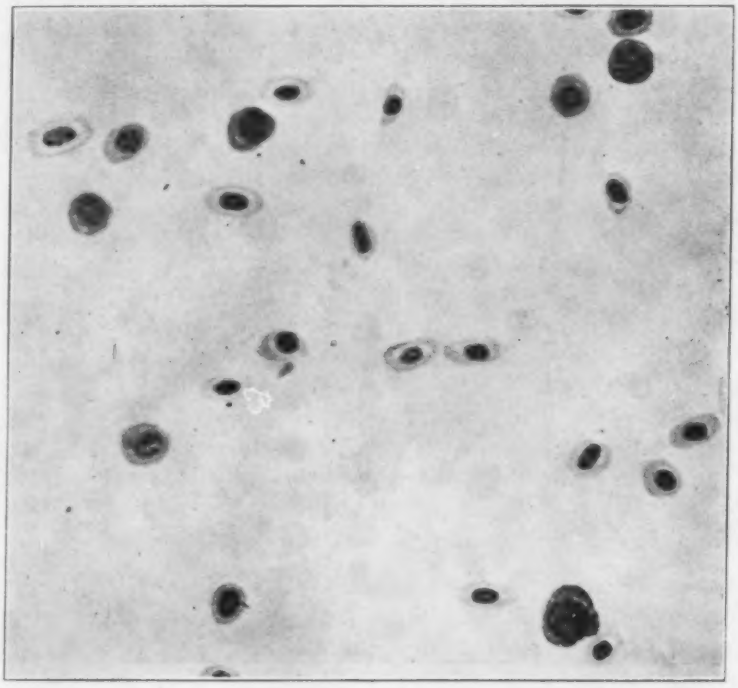

FIG. 6. Blood picture of case V462. Case diagnosed as erythroleukosis.

type. However, considering the existing controversy concerning its relationship to fowl leukosis and the large number of cases in which the nerve lesions are the only ones noted, a special classification for them based on location only seems justified.

The lesions in the nerve type have been noted in practically all of the main peripheral nerve trunks, brain, spinal cord, ganglia and cranial nerves. Probably the most common locations are in the ischiatic plexus and nerve, brachial plexus and vagus nerve, though they have been quite frequently noted in

the cervical, visceral and intercostal nerves. Affected nerves lose their normal, white, glistening appearance. They are enlarged and of a dull gray or yellowish gray color. The irregular striations become obscure and in badly affected nerves completely disappear. The affected nerves generally show a diffuse, lymphoid-type tissue infiltration or hyperplasia, though a tendency toward distinct nodular formations is noted, particularly in the vagus nerves and on the floor of the cranial cavity

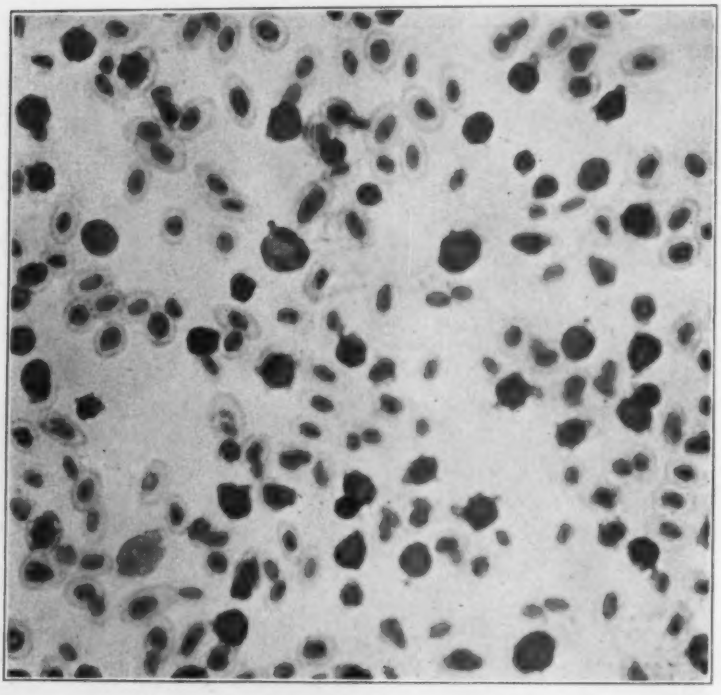

FIG. 7. Another blood picture from case V462.

originating in the nerve roots of that region. These cranial nodules are found frequently in birds affected with leukosis that clinically show various deviations of the head, at times to such an extent that the back of the head rests on the floor or ground. Marked variations are shown in different birds in regard to location of lesions, extent and degree of involvement. The clinical symptoms naturally depend upon the location of the nerves involved.

The eye type presents a very interesting study. This type is chiefly characterized by a depigmentation of the iris, con-

striction and malformation of the pupils, and impaired vision. The color of the iris changes to a gray, slate or bluish color. Complete blindness is sometimes noted. The lesions in the eye type are principally inflammatory in character, though melanotic neoplastic formations are noted occasionally in the iris. Histologically the predominating cells are of the eosinophile type in some cases, while they are apparently undifferentiated lymphoid-like cells in others. The lesions vary much in different cases in regard to degree and extent of involvement.

The blood picture in uncomplicated eye-type cases is very similar to those described in the nerve type. However, this type is frequently associated with the erythroid and myeloid types. In such cases the blood pictures are of the nature of those described under those types. The eye type also is very frequently noted in combination with the nerve and lymphoid types.

Mixed types: Practically any conceivable combination of types may be noted. Many cases, apparently showing only one type in the beginning, often develop other types also after varying periods of time. This has been noted particularly in original eye-type cases. Perhaps this is because the eye-type cases are so apparent clinically and birds showing uncomplicated eye-type lesions generally remain healthy otherwise for a long time, usually as long as vision is not too greatly impaired. However, a very large percentage of these cases, when kept for breeding purposes and for observation, developed one or more of the other types after variable periods of time.

Our pathological studies indicate that all the different types discussed are different expressions of the same disease.

TRANSMISSION

Our studies on the transmission of fowl leukosis extend over a period of four breeding seasons. Comparatively large numbers of chickens were used. These experiments include injections of suspensions made from material obtained from different types of fowl leukosis. Filtrates have also been used. Studies on the transmission by direct and indirect pen contact and on the transmission through the egg have been made. Chicks have been reared on contaminated litter to determine its infectiveness. Studies which include different strains of chickens and pedigree breeding to determine the hereditary influences involved were made. The results of these studies are briefly summarized as follows:

Fowl leukosis in all of its expressions was transmitted by the injection of suspensions prepared from fresh leukosis ma-

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terial, by direct and indirect pen contact and by rearing chicks on infective litter. All types of fowl leukosis described in this paper are caused by the same etiologic agent. However, different strains of chickens may show variations in regard to the predominant type produced. Different chickens of the same strain also show wide differences in types produced by the same strain of virus. The above findings clearly indicate that the individual fowl and strain of fowls used are important contributing factors in the types produced, though exposure to one type has produced practically all types in chickens of the same strain. These observations were noted both in chickens in which the disease was produced experimentally and in chickens in spontaneously affected field flocks. Our studies show also that there is a marked difference in the degree of resistence and susceptibility between chickens of the same strain and between different strains of chickens as a whole; also, that these factors are inherited. Our studies on the direct transmission through the egg indicate that the great majority of field cases are not produced in this manner but by direct contact with the etiologic agent after the chicks are hatched. It is very doubtful if the disease is often, if ever, transmitted directly through the egg. Evidence thus far obtained strongly suggests that the etiologic agent is of the nature of a filtrable virus.

Control

The most important factors in the control of fowl leukosis in the present state of our knowledge seem to be the following:

Eliminate affected birds as soon as noted and attempt to build up resistant strains by the process of elimination and proper selection of breeding stock. When new breeding stock is introduced, secure them from strains that are highly resistant to fowl leukosis as far as possible. Rear chicks on clean ground away from other poultry. Rigidly enforce proper hygiene and sanitation.

Discussion

The views expressed in this paper are by no means generally accepted by all investigators of fowl leukosis. Opinions vary widely in regard to the relationship between the different types described in this paper. However, the gap seems to be gradually closing in, and it is hoped that there will soon be a more uniform agreement.

Summary

The material used as the basis for this paper includes studies on over 2,000 chickens. Approximately 850 were diagnosed as

positive fowl leukosis cases. Although the great majority of the material was, as already stated, obtained from our investigations at Iowa State College, some of it was derived from our clinical and pathology studies made at the Alabama Polytechnic Institute in the College of Veterinary Medicine.

The pathology of the different types has been described and a brief summary of our various transmission experiments given. Hereditary influences also have been briefly discussed.

Conclusions

Our studies indicate beyond a reasonable doubt, that the myeloid, erythroid, lymphoid, nerve and eye types of fowl leukosis, as discussed in this paper, are different expressions of the same transmissible disease; that the neoplastic lesions variously termed round-cell sarcoma, lymphosarcoma, lymphatic leukemia, and aleukemic and leukemic lymphocytoma are fundamentally the same as lymphoid leukosis; that the disease in all of its expressions can be transmitted by the injection of suspensions prepared from fresh fowl leukosis material, by direct and indirect pen contact, and by rearing susceptible chicks on infective litter; that inheritance plays a very important rôle in regard to the degree of resistance and susceptibility of chickens to fowl leukosis. These hereditary factors are of great importance in the control of and studies of fowl leukosis.

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Discussion

Dr. M. W. Emmel: Doctor Patterson spoke of different strains being more susceptible than others. We have just conducted some experiments in which we induced the disease by microörganisms. We secured twelve strains of chicks, 25 in each group, from a number of southern states. We raised the birds alike, and marked them so that we knew their origin. Then they were placed in cages, so that they were all mixed. We could not tell which bird was which until we took it out of the cage and looked at the band.

The experiment was conducted first on White Leghorns and repeated later on Rhode Island Reds. In the most susceptible group of White Leghorns, 60 per cent came down with one of the pathological conditions. In the best group, only 5 per cent came down. Comparable results were encountered in the case of Rhode Island Reds. This would indicate that there is considerable difference in strains of chickens as

far as susceptibility is concerned, regardless of whether they are one breed or another.

DR. PATTERSON: There was no intention, on my part, to leave the impression that Rhode Island Reds, as a breed, are less susceptible to fowl leukosis than are White Leghorns. It was my purpose to bring out the fact that different strains of chickens, regardless of breed or variety, show marked differences in regard to their resistance and susceptibility to fowl leukosis, and that these factors are inherited. It so happened that the particular strain of Leghorns referred to in our paper was much more susceptible to fowl leukosis than was the particular strain of Rhode Island Reds used by us to illustrate these differences.

Research on Wildlife

The Bureau of Biological Survey of the United States Department of Agriculture has selected eight states in which to conduct coöperative research on game. The states which have been designated for this work are: Virginia, Oregon, Texas, Iowa, Maine, Connecticut, Alabama and Utah. In each state the Agricultural College and the State Conservation Department will coöperate with the United States Bureau of Biological Survey, according to Dr. J. E. Shillinger, in charge of disease control, Division of Wildlife Research, Bureau of Biological Survey.

New York Membership in the A. V. M. A.

Year	*Members*	*Year*	*Members*
1923	224	1929	263
1924	230	1930	261
1925	225	1931	270
1926	214	1932	259
1927	232	1933	255
1928	241	1934	250
1935	267		

Egg Production Increasing

The number of hens and pullets of laying age in farm flocks averaged 65.4 per farm on October 1, 1935, compared with 64.5 on the same date in 1934, according to the U. S. Department of Agriculture. Layings per farm flock averaged 16.7 eggs on October 1, compared with 15.7 eggs one year previously. Both figures are below the 1928-1932 average, which was 71.4 for the number of hens and pullets, and 18.1 eggs per farm flock.

HEMOCYTOBLASTOSIS AND ITS RELATION TO THE DEVELOPMENT OF FOWL PARALYSIS AND FOWL LEUKEMIA*

By M. W. EMMEL, *Gainesville, Florida*
Florida Agricultural Experiment Station

The term hemocytoblastosis was first used in connection with the development of fowl paralysis and leukemia by Johnson[1] to designate the presence of hemocytoblasts in the peripheral circulation and a proliferation of these cells in the bone-marrow. Johnson observed that hemocytoblasts and lymphocytes may infiltrate the central nervous system, peripheral nerve trunks and visceral organs, resulting in the development of the lesions found in fowl paralysis, leukemia and allied conditions in the fowl.

Johnson contends that the cause of hemocytoblastosis is a "filtrable agent" and claims this agent a primary cause of fowl paralysis and leukemia, although the major portion of his investigations dealt with the transmission of these diseases by means of the injection of tissue emulsions and filtrates of affected organs into susceptible birds.

Patterson, Wilcke, Murray and Henderson[2] had previously concluded that fowl paralysis and leukemia were caused by a common agent, perhaps a filtrable virus; they failed, however, to observe hemocytoblastosis.

Previous papers[3-6] have indicated that fowl paralysis, leukemia and allied conditions in the fowl are caused by a common agent, certain species of the paratyphoid and typhoid groups of bacteria, and that certain factors influence infection. These factors also influence, in a large measure, the development of the pathological manifestation which results from the infection. "Light," anemia, fowl paralysis, lymphomatosis, leukemic lymphocytoma, aleukemic lymphocytoma, myeloid leukosis, erythroleukosis and various combinations of these pathological processes may result from infection by a specific microörganism. Hemocytoblastosis has resulted from oral exposure and intravenous injection of the causal microörganisms and is present during the incubation period of the disease with the possible exception of aleukemic lymphocytoma.

These studies have revealed also that cases of erythroleukosis or myeloid leukosis induced by the causal microörganisms may then be transmitted to healthy birds by the injection of tissue emulsions and filtrates of affected organs after the technic of

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Johnson, Patterson *et al*, and the numerous other investigators who have conducted transmission experiments. It is interesting to note that hemocytoblastosis was an essential process in the transmission of leukemia in these studies. These investigations confirm the observations of Johnson that hemocytoblastosis is a fundamental process in the development of fowl paralysis and leukemia, but indicates that original cases of the disease are induced by microörganisms of the paratyphoid and typhoid groups. Johnson failed to give convincing data as to the natural avenue of infection with a "filtrable agent" as a primary cause of this group of diseases.

Hemocytoblastosis, being of such a fundamental nature in the development of fowl paralysis and leukemia, is worthy of considerable study. The term hemocytoblastosis, however, fails to describe the blood picture completely. During this process a variable number of degenerative blood cells are present in the peripheral circulation. Warthin[7] was the first investigator to comment upon this fact, which has received but little attention from numerous subsequent investigators.

The Role of Enteritis

A preliminary report[1] on this investigation indicated that enteritis, most commonly caused by intestinal parasites, was necessary, so that an avenue of infection would be created for the causal microörganisms and in order that the infection result in one of this group of diseases. A subsequent paper[4] somewhat modified this statement, as it was found that a particularly virulent strain of one of the causal microörganisms had the ability to penetrate the intestinal mucosa without the aid of enteritis, with the subsequent development of fowl paralysis or leukemia. This is particularly true experimentally when dealing with chickens under six weeks of age. Cases developing under such circumstances are not numerous. Large numbers of birds, however, develop hemocytoblastosis experimentally following oral exposure to the causal microörganisms but with no or very little previous inflammation of the intestinal tract. In most of these cases, the impetus given hemocytoblastosis is insufficient for the process to progress under its own stimulus as is the case in instances in which infection following enteritis occurs. These investigations indicate that enteritis is a necessary predisposing factor when an outbreak of fowl paralysis or leukemia of consequence develops.

Johnson's experiments showed that birds often recover from hemocytoblastosis when exposed in transmission experiments.

Results in this investigation have shown that birds may recover from hemocytoblastosis caused by the primary causal microörganisms and also when exposed to the transmission agent. With the exception of myeloid leukosis and erythroleukosis the course taken by hemocytoblastosis offers little evidence as to which pathological manifestation may develop. Birds often recover from severe cases of hemocytoblastosis, males more often than females. Recovery results in considerable immunity both to the causal microörganism and to the transmitting agent. If birds are exposed either to the causal microörganism or transmitting agent during active hemocytoblastosis, an increased impetus to the process results with more cases of fowl paralysis and leukemia developing and often with a considerable shortening of the incubation period.

Hemocytoblastosis may develop in a flock from light infection by the causal microörganisms, with the birds showing no clin-

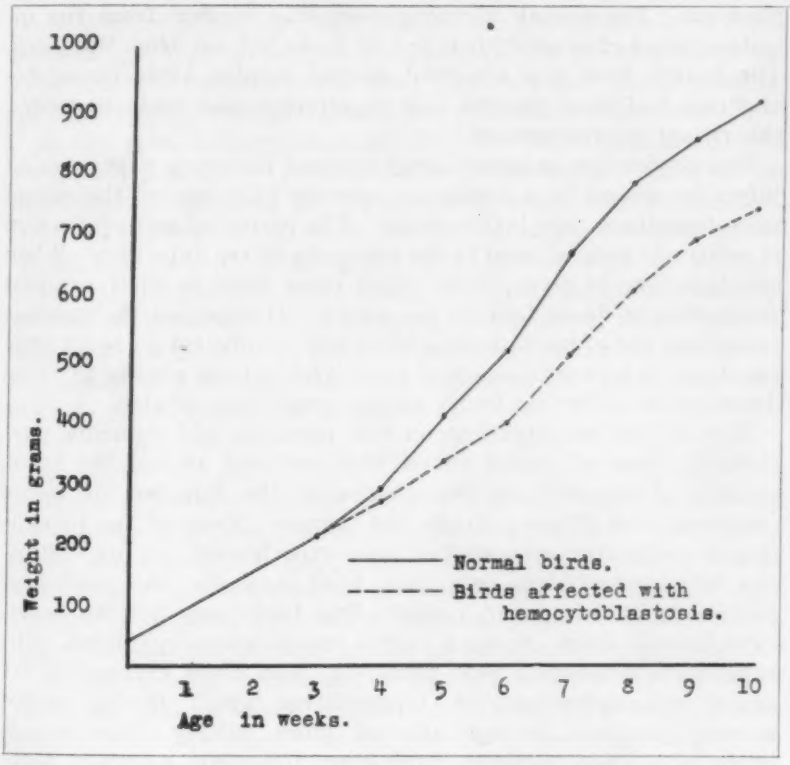

FIG. 1. The growth curve of 10 normal Barred Rock chickens and 10 Barred Rock chickens (5 males and 5 females in each group) affected with hemocytoblastosis caused by the injection of a suspension of *S. aertrycke* when three weeks of age.

ical symptoms other than retarded growth or decreased egg-production. The graph in figure 1 shows the growth curve of two groups of Barred Rock chickens (five male and five females in each group), one group infected by a single intravenous injection of *Salmonella aertrycke* when three weeks of age, while the other group served as a control. Hemocytoblastosis resulted from the infection in each of the ten birds exposed and all recovered.

Disease Diagnosed in Four Flocks

During the past two years, hemocytoblastosis has been diagnosed in four flocks of laying birds. Mortality was not unusual, the combs and wattles of many birds were white and scaly on the surface but not particularly anemic, no cases of paralysis had occurred, and egg-production decreased from a satisfactory percentage to less than 15 per cent, and in one instance to 3 per cent. Decreased production was experienced for a period of three to four months. Blood smears indicated the presence of hemocytoblastosis. The causal microörganism was isolated from the intestinal tract of several birds in two flocks but not from the third. The fourth flock was observed several months after hemocytoblastosis had been present and no attempt was made to isolate the causal microörganism.

Egg-production is immediately reduced following experimental infection caused by a single intravenous injection of the causal microörganisms into laying birds. The period of non-production is relatively proportional to the intensity of the infection. When one injection is given, birds often come back to their original production in from four to six weeks. If repeated intravenous injections are given to laying birds but insufficient to cause fowl paralysis or leukemia, cases of hemocytoblastosis similar to those described as occurring under natural conditions develop.

Most of the investigations on fowl paralysis and leukemia, particularly those of recent years, have centered around the agent capable of transmitting the disease by the injection of tissue emulsions and filtrates of affected organs. Many of the investigators conducting such studies have experienced varying difficulties with control birds developing fowl paralysis. As mentioned previously in this paper, parasite-free birds may develop hemocytoblastosis from exposure to the causal microörganisms, with some birds developing fowl paralysis; also, birds exposed to the causal microörganisms or transmitting agent during active hemocytoblastosis become affected more quickly than normal birds and when exposed during or following recovery from hemocytoblastosis considerable immunity is encountered. These facts are of the utmost importance when investigations are being

conducted on fowl paralysis and leukemia. Failure to give these facts consideration will lead to inaccurate results, with consequent erroneous interpretations.

The plant used in the course of these investigations was so arranged that birds were raised from baby chicks, in a system of batteries in a room more or less isolated but in close proximity to infected birds on experiment. The two doors were provided with a tray for the disinfection of the shoes on entering. All feed was kept separate from that of other birds. The equipment was disinfected thoroughly once a week and the floors and walls of the room twice a week. Water-troughs were cleaned and filled once a day, and disinfected twice a week. The same attendant, however, cared for all of the birds. This plan proved satisfactory for ten months, but since that time, in spite of various modifications that have been made, it has been impossible to raise birds free of hemocytoblastosis. This has necessitated the raising of birds for experiment purposes at a location far removed from infected birds. In other words, two plants have been found necessary, one for experiments in which birds are exposed to the causal microörganisms could easily be introduced in this manner. used for éxperiments are raised free of hemocytoblastosis.

In the past, bacteria have received but little consideration as the primary cause of fowl paralysis or leukemia. Many of the investigators carrying on transmission experiments have held laboratory postmortem examinations on large numbers of infected birds and it is quite logical to believe that the primary causal microörganisms could easily be introduced in this manner. In view of these facts it is necessary, in order to obtain reliable data, that all birds be examined for hemocytoblastosis before being placed on any experiment pertaining to fowl paralysis, leukemia or allied conditions in the fowl.

SUMMARY

Hemocytoblastosis can be induced by oral exposure and the intravenous injection of microörganisms of the paratyphoid group and by the intraperitoneal injection of tissue emulsions and filtrates of the organs of affected birds. During hemocytoblastosis the blood cells show various types of degeneration. Hemocytoblastosis induced by the causal microörganisms or the transmitting agent may lack the impetus necessary to the development of fowl paralysis or leukemia. Young birds affected with hemocytoblastosis show retarded growth, while laying birds show decreased egg-production. Birds used in experiments must be hemocytoblastosis-free to insure obtaining reliable data.

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Discussion

Dr. A. J. Durant: In the early stages of hemocytoblastosis, were there any gross pathological changes observed in the organs?

Dr. Emmel: Not immediately. Within a very short time, after exposure of the birds to the causal organisms, we get the blood picture of hemocytoblastosis. The length of time that intervenes between the time that hemocytoblastosis first appears and the time that microscopic lesions are first noted is variable. For instance, in anemia and emaciation, the microscopic lesions in those cases appear very quickly. In fowl paralysis, it takes just a little bit longer, and in the case of leukemia, it takes still longer, so that factor varies considerably; but the first change that we notice is in the bone-marrow. We get a degeneration of bone-marrow, possibly because of the fact that the bone-marrow is trying to replace degenerated blood cells in the circulation.

Dr. Durant: In birds that die of hemocytoblastosis, what are the gross changes?

Dr. Emmel: As a rule, birds do not die from what we call hemocytoblastosis. They may develop some of the other pathological conditions as a result of hemocytoblastosis. We have had many cases in which birds have recovered from hemocytoblastosis. We have never failed to produce a case of hemocytoblastosis by exposure to the causal organisms, but there are a number of factors which influence infection, and make the picture so variable that it is very difficult to set any hard and fast rules as to just what will happen after these organisms disappear from the picture.

I might say that we have isolated, in a rather crude form, the substance which is responsible for the continuance of hemocytoblastosis, after the causal organisms have disappeared. We have attempted, in the chicken, to produce an antiserum against that particular substance. We have not as yet produced an antiserum which is as potent as I would like to get it, but the antiserum which we produced on the first attempt has cured every chicken we have injected it into, provided it was an uncomplicated case of hemocytoblastosis. Those cases that were complicated died. In the case of other animals, the blood picture following the injection of the causal organisms is considerably different from that in the chicken.

Dr. Durant: What is the causal agent?

Dr. Emmel: We refer to microörganisms of the paratyphoid and typhoid groups as the causal agents. When we speak of the transmitting agent, we refer to the tissue emulsions and filtrates of affected organs by which many investigators have shown that leukemia can be transmitted.

ATTEMPTS TO ISOLATE BRUCELLA ABORTUS FROM THE BLOOD OF DOGS IN EXPERIMENTAL BRUCELLOSIS*

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This work represents a study of bacteriemia in dogs with experimental brucellosis and of attempts to induce a bacteriemic state by means of histamine and anaphylactic shock.

Bacteriemia, in brucellosis of various species of animals, is not constant. The isolation of *Brucella abortus* from the blood of a human being suspected of having undulant fever affords a conclusive laboratory test for the diagnosis of the illness.

Soulé[1] made two series of tests, approximately six months apart, on 5,000 cows. The first test revealed Brucella agglutinins in the blood of 2,239 cows, of which 299 gave positive blood cultures for Brucella. In the second series, agglutinins were present in the blood of 2,607 animals, and 206 gave positive blood cultures. He also obtained positive blood cultures in 40 animals when agglutinins were not demonstrable. Fitch, Bishop and Boyd[2] seldom found Brucella present in the blood of infected cattle. They obtained 123 samples of blood from 15 cows that were positive to the agglutination test, and from four animals that were artificially infected. These samples were collected before, and up to 47 days after, the end of gestation. Only four samples yielded the organism, and two of these were from the same animal.

From artificially infected swine, Cotton and Buck[3] recovered the organism from the blood from the tenth to the 45th day after inoculation. From five of the twelve swine used, the organism was obtained but once; from three of the animals, twice, and from four of the animals, four times. Each animal was tested for bacteriemia from four to ten times.

Henry, Traum and Haring[4] obtained Brucella from the heart-blood of experimentally inoculated guinea pigs in 47.6 per cent of 123 infected animals.

Feldman, Bollman and Olson[5] found that five of eleven dogs gave positive blood cultures after intravenous or oral inoculation with the suis variety of Brucella. One dog gave a positive culture 20 days after inoculation. One animal died, apparently as a result of brucellosis, 39 days after inoculation and the organism

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was isolated from the heart-blood. With these exceptions, successful isolations were obtained either ten or 14 days after inoculation. Van der Hoeden[6] made six attempts, with three successful isolations of the organism from the blood of 13 experimentally infected dogs.

METHODS

Five dogs were used as test animals. According to Manwaring and his coworkers,[7] dogs may be sensitized to horse serum so that about 95 per cent of them react to the shocking dose.

Three of the dogs (1, 2 and 3) were injected subcutaneously with 0.5 cc of horse serum per kilogram of body weight, and a like quantity was given intravenously 24 hours later. Two of these animals (1 and 2) 21 days later received intravenous injections of 1 cc of horse serum per kilogram to produce shock. Two dogs (4 and 5), not previously sensitized to horse serum, received intravenously 2 mg of histamine per kilogram of body weight. Dog 3 was not injected with either horse serum or histamine to produce shock.

One cc of a dense saline suspension of a suis strain of *Br. abortus* (our number 1363) was given intravenously to all five dogs. This strain was isolated 28 months previously from a lesion of lumbar spondylitis in a hog, and the organism has been used in various experiments and its pathogenicity for the guinea pig has varied but little since its isolation. The inoculation was received by the dogs 16 days before the shocking dose of horse serum or the administration of histamine.

Blood cultures and tests for Brucella agglutinins were made before inoculation with *Br. abortus*, and six hours, one day, three days, one week, two weeks, and 15 days, respectively, after inoculation. They were also made before the shocking dose of horse serum or histamine, and ten minutes, 30 minutes, one hour, six hours, and three, five, eleven, and 13 days, respectively, after the animals had received the shocking dose of horse serum and histamine. Blood (10 to 15 cc) was obtained from the jugular vein. Ten cc of blood was placed in a sterile tube with a small amount of 20 per cent solution of sodium citrate and was evenly distributed among six Petri dishes to which was then added potato-agar medium. The plates were incubated at 37.5° C. for from four to five days. Colonies suspected of being *Br. abortus* were subcultured and identification was made by tests for specific agglutination. The serums for agglutination tests were secured by placing 5 cc of blood in a Kolmer tube and the serum was harvested from the resultant clot. The antigen used for aggluti-

nation was composed of five strains of *Br. abortus*, suis variety, and one goat strain.

Splenic biopsy was made on all of the dogs 39 days after inoculation for culture. Approximately 10 gm of tissue was removed from each dog. This sample was ground in a sterile mortar, with sterile normal salt solution. Portions of the supernatant fluid then were cultured and two guinea pigs were inoculated with the material from each portion of spleen.

Results and Comment

All dogs yielded negative blood cultures before inoculation.

Br. abortus was recovered from the blood of each of the dogs at least once during the 14 days after inoculation. At only one period was bacteriemia of all dogs manifest at the same time; namely, 24 hours after inoculation. Three dogs (1, 2 and 3) yielded positive blood cultures 48 hours after inoculation. At the three-day and 15-day intervals, all dogs failed to reveal bacteria in the blood. Six hours and seven days after inoculation, only dog 4 was positive. Fourteen days after inoculation, only dog 3 was positive.

Only once was a positive blood culture secured after histamine shock; this was in dog 4, ten minutes after the animal had received histamine. All attempts to isolate *Br. abortus* from the blood of dogs presumed to be sensitized after receiving the shocking dose of horse serum failed.

The agglutinative titres reached their maximal height 14 days after the animals had received the organisms. The titres fluctuated somewhat after histamine shock and the last injection of horse serum, but the tendency was to drop with the passage of time. It is of interest that from dogs 1, 3 and 4 the organisms were isolated from the blood-stream before an agglutinative titre of 1:12 could be demonstrated. In dogs 2 and 5, a low titre was found (1:12 and 1:25, respectively) before inoculation with *Br. abortus*. This titre was not increased at the time successful isolations were first made from the blood 24 hours after inoculation. The interval was not long enough for a demonstrable rise of agglutinins to take place.

Both dogs that received histamine developed a marked anaphylactoid reaction. Contraction of the urinary bladder, relaxation of the sphincters, and dyspnea occurred. The two dogs which received the horse serum after being previously sensitized gave no clinical evidence of shock. Although these animals manifested no symptoms, it is possible that there was some systemic disturbance as a result of the shocking dose of horse serum. Certain

experiments are contemplated in which some index of the reaction to horse serum in previously sensitized dogs may afford a means of repeating this portion of the experiment in a more suitable way.

Splenic biopsy 39 days after inoculation with Brucella made it possible to demonstrate that the animals were harboring the bacteria within their tissues during the experimental period. Direct culture of the splenic tissues and inoculation of guinea pigs with this material yielded Brucella in every instance. There were no gross pathologic alterations in the spleens at the time of biopsy.

SUMMARY

In each of five dogs that were intravenously inoculated with *Brucella abortus* of swine origin, bacteriemia was demonstrated rather constantly within the first 48 hours following inoculation.

Two of the five animals we attempted to sensitize to horse serum failed to give clinical evidence of anaphylactic shock when a shocking dose of horse serum was administered. Bacteriemia was not demonstrable in either of these two after they had received the dose of horse serum which was intended to produce shock.

In two other dogs of the group, anaphylactoid shock was produced by histamine. The single isolation of Brucella from the blood of only one of these two animals indicates that this mechanism is probably not closely associated with the induction of bacteriemia in experimental brucellosis of the dog.

The presence of Brucella organisms in the tissues of the various dogs during the course of the experiment was ascertained by splenic biopsy 39 days after inoculation. The organisms were isolated from each dog at that time.

There was no significant disturbance in the agglutinative titre of the serums of two dogs after histamine shock.

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THE TOXICITY OF BROOMWEED (GUTIERREZIA MICROCEPHALA) FOR SHEEP, CATTLE AND GOATS*

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For years ranchers have suspected that the broomweeds were poisonous for cattle and sheep, and the results of investigations of certain live stock losses have, to some extent, supported the ranchers' opinions. However, in feeding tests with two species of perennial broomweeds, Schmidt[1] and Boughton[2] failed to obtain toxic effects. Continued field observations suggested the necessity of further investigation of broomweeds, and during the spring and summer of 1933 feeding tests of *Gutierrezia microcephala* were undertaken. This investigation produced negative results, for which no explanation is available at this time. In spite of the negative findings, the work was repeated during the spring and summer of 1934 with quite different results.

BOTANICAL DESCRIPTION‡

Gutierrezia microcephala (DC.) A. Gray is a low (12 inches or so in height) corymbosely-branched perennial herbaceous plant having a woody base with one to three short, leafless stems which bear slender, leafy branches that terminate in a flat-topped flower cluster of small, yellow-flowered heads. Each stem may bear from a few to as many as ten or 15 of these slender, ascending branches, and a well-developed plant of 20 or more branches may bear about 2,000 heads. The branches are circular in cross-section, angled or ridged, free from hair, leafy to the base and terminate in very slender flower branches arising from all along the two or three inches of the apical end of the branch.

In times of drought or at maturity the lower leaves die and drop off, while the remaining leaves stay green. The foliage is of numerous, alternate, thread-like leaves, which are less than a millimeter wide and from 10 to 45 mm long; they are covered with a sticky exudate and taste something like turpentine. The heads are small and numerous, usually short-stalked at the end of the thread-like flowering branches. The stalks are usually less than 8 mm. long, but of varying lengths and usually with three heads in a cluster. The flowering branches are of varying

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‡Botanical description by V. L. Cory, Texas Agricultural Experiment Station, Sonora, Texas.

lengths, being progressively shorter as the apex is approached. The very tip of the flowering branch bears a cluster of 2 or 3 heads that are not stalked, or sometimes very shortly stalked. The effect is to form a flat-topped cluster of heads on each branch and thus for the plant as a whole.

The involucre is top-shaped, about 3 mm long and 1.5 mm broad with leathery bracts in three or four series, but few in number. The bracts are whitish, sticky, and all but the inner-

FIG. 1. *Gutierrezia microcephala.*

most have greenish or herbaceous tips. The involucre contains from 3 to 6 each of ray-flowers and of disk flowers, both having yellow corollas. The flattened corolla of the ray-flower is fully three-fourths the length of the involucre, or as much as 2.5 mm long. The achenes of the disk are less than 1 mm long, frequently about half that length, broad at the apex and wedge-shaped to the acute base. The pappus scales, about 6 in number, are narrow and about twice the length of the achene, or more than 1 mm long. The achenes of the ray are 1 mm long, linear-oblong, or slightly broader at the apex, and with a pappus of shorter,

broader, rounded scales, less than half the length of the achenes, or less than 0.5 mm long.

This species extends southward into northern Mexico, and from the best information available, it occurs as far west as Idaho and Arizona. It extends from these states through New Mexico to well across Texas, the easternmost collection, so far as known, being from Grimes County, Texas, and just west of the East Texas timber belt. From this point its northern line of occurrence in Texas extends northwesterly to include much of the Texas Panhandle.

Experimental Feeding

In all tests the leaves and about three inches of the tip of the branches were fed, the woody portion of the stems and branches being excluded. The plant was collected within a radius of ten miles of Alpine. The green plant was fed to all animals except in the case of sheep 52 and 54, which were fed the dry plant. The tests were started as soon as the first growth was obtainable in the spring and continued into the summer when the plant was in full bloom. With one exception force-feeding was required, as the animals refused to eat their allotted amounts. Alfalfa hay

Table I—*The feeding of G. microcephala to sheep, cattle and one goat.*

Animal	Weight (Lbs.)	Plant Fed (Lbs.)	Feeding Period (Days)	Results
Sheep				
44*	85	10 green	5	Hematuria. Died
45*	85	8 green	5	Hematuria. Later recovered
46†	83	8 green	5	Hematuria. Died
50*	80	8 green	9	Nephritis; no hematuria. Recovered
52‡	70	2.2 dry	4	Nephritis. Died
54‡	110	10 dry	63	No ill effects
Cow				
92*	400	24 green	3	Nephritis. Died
78†	700	101 green	47	No serious results
Goat				
86†	60	11 green	14	Nephritis. Died

*Fed young plant.
†Fed older plant.
‡Fed plant in blooming stage.

was fed *ad libitum* in addition to the broomweed. The results of feeding six sheep, two cattle and one goat are summarized in table I.

The poisonous nature of this plant was demonstrated by the fact that as little as eight pounds, when fed within a period of

five to nine days, was sufficient to produce serious illness in some sheep and death in others. The feeding of 2.2 pounds of the dry plant (equivalent to 8.8 pounds of the green plant), during a period of four days, was sufficient to cause the death of sheep 52. Fatal poisoning was produced in steer 92 as a result of feeding eight pounds of green plant per day for three consecutive days, or a total of 24 pounds, death occurring five days after the administration of the last dose.

In the case of the goat, 11 pounds of the plant were sufficient to produce fatal results in 14 days. Small doses of the plant were tolerated without toxic effects, as sheep 54 consumed 10 pounds of the dry plant during a feeding period of 63 days without ill effects. Cow 78, a pregnant animal, was fed 101 pounds of the green plant during a period of 47 days without ill effects. Three different stages of growth of the plant were fed to the sheep and since the results in the case of sheep 52, which was fed the blooming plant, are similar to the results obtained when the younger plant was fed to other sheep, it is evident that the toxic content did not decrease to any extent as the plant approached maturity.

Clinical Manifestations

A loss of appetite, the first evidence of poisoning, was soon followed by a listless attitude, arched back and drooping head. In severe cases pronounced hematuria occurred within a day or two after the appearance of the first symptoms. The presence of hematuria was evidently governed by the size of the individual dose as this condition was observed only in the sheep which received the largest daily doses. In the milder cases of poisoning, nephritis without hematuria was observed. A well-marked icterus of the sclerotic coat of the eyeball was a constant finding, but this change could hardly be detected in the mucous membranes.

Blood studies revealed changes which were about what one would expect in view of the nephritis. As a result of the hematuria in the severe cases there was a decrease in the red blood corpuscle content of the blood. There was a mild leukocytosis which was evidently due to a marked increase of polymorphonuclear leukocytes. The non-protein nitrogen content of the blood was found to be extremely high in some cases (130 mg per 100 cc of blood).

Pathology

The five cases which died were autopsied for gross and microscopic study. Upon autopsy the icterus which was first detected

in the sclera was observed also in the tendinous portions of the body but not in the muscles or skin. The kidneys were very much swollen in all cases. In the most severe form of poisoning numerous hemorrhages were observed in the cortex; in the milder cases no hemorrhages occurred, but a yellowish-brown color of these organs was suggestive of considerable change.

FIG. 2. Photomicrograph of a section from a kidney showing hemorrhage into Bowman's capsules and tubules. Also considerable degeneration of the tubular epithelium.

The liver exhibited a yellowish mottled appearance, the color changing to green upon fixation in formaldehyde. No other gross lesions were observed.

The microscopic changes in the kidneys of acute cases consisted of hemorrhage, albuminous degeneration, and necrosis. In many fields the Bowman's capsules, as well as many of the tubular structures, were distended with blood. Interstitial hemorrhages were of frequent occurrence and in some cases these hemorrhages could be traced from the connective tissue into the lumen of tubules. There was an albuminous degeneration and early necrosis of the epithelium of the convoluted portion of the tubules, but the retrograde changes were less marked in the straight and collecting tubules. In less acute cases no

hemorrhages were observed but there was pronounced necrosis of the epithelium of the convoluted portion of the tubules. Here again the epithelium of the straight and collecting tubules appeared to have escaped much of the destructive action of the toxic principle.

The liver showed albuminous degeneration, but the essential lesion was one of liquefaction necrosis. The pyknosis of the

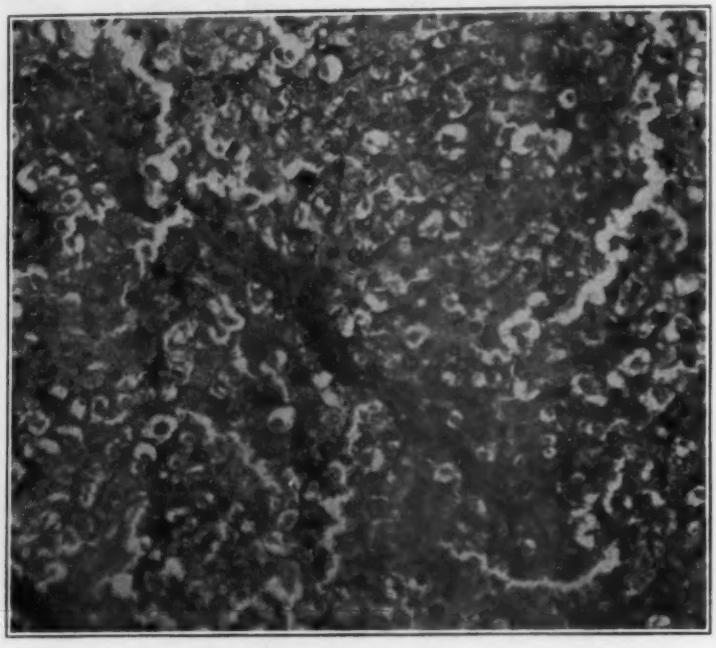

FIG. 3. Photomicrograph of a section from a liver showing liquefaction necrosis of the liver cells.

nucleus was soon followed by the appearance of a liquefied band around the periphery of the cell. As the necrosis approached the central portion of the cell the nucleus gradually lost its staining power but did not undergo karyorrhexis. The predominating picture was one in which a shrunken portion of cytoplasm containing a faintly stained nucleus was surrounded by a liquefied band; however, complete liquefaction was frequently observed.

DISCUSSION

This broomweed may be grazed by sheep during the winter, in the presence of fair range conditions, if climatic conditions have been favorable for the production of new growth of the plant

but no other green forage. Otherwise the plant becomes of importance as a source of live stock loss only under unfavorable range conditions, which is especially true for cattle. We have not observed a serious outbreak of this form of poisoning, although it is probable that such has occurred without having been called to our attention. Nevertheless the losses, though sporadic, must have amounted to a rather large total, especially during the period of drouth which has prevailed for some time over the greater part of the Southwest.

It is common opinion among ranchers of the Southwest that this plant is responsible for abortions in cattle. We have failed to produce abortion by feeding the plant to one cow and a sheep which was fed to the point of prostration, later recovered and gave birth to a healthy, live lamb. Such results are too limited to warrant any conclusions as to the abortifacient properties of *G. microcephala* and will have to be augmented by other experiments before definite conclusions can be drawn.

SUMMARY

1. *Gutierrezia microcephala* was proven to be toxic for sheep, cattle and goats.

2. Fatal results were produced in sheep by feeding eight pounds of the plant during a period of five days, in a steer by feeding 24 pounds in three days, and in a goat by feeding eleven pounds in 14 days.

3. The lesions were confined to the kidneys and liver. In acute cases there were hemorrhages into Bowman's capsules and into the interstitial connective tissue. In milder cases there was no hemorrhage but pronounced necrosis of the epithelium of the convoluted portions of the tubules. The histopathology of the liver was found to be liquefaction necrosis.

4. As a rule losses are sporadic and occur as a result of unfavorable range conditions.

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Purebred Guernseys to Alaska

A herd of 55 purebred Guernsey cows were shipped to Alaska the past month, to be distributed among families in the Matanuska colony. The distribution of the cattle will be by lot, one cow to a family. Dr. J. B. Loftus (Colo. '29), of the University of Alaska, selected the herd. He is of the opinion that Guernseys will prove adaptable to the northern climate and feed.

DOES THE RIGHT OVARY OF THE BOVINE FUNCTION MORE FREQUENTLY THAN THE LEFT?*

By C. F. CLARK, *East Lansing, Mich.*
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It is of interest that the right ovary of the bovine is said to function more frequently than the left. Hammond,[1] Dukes,[2] Somer,[3] Stoss[4] and Tutt[5] have stated this belief and Schmaltz[6] has gone as far as to state that left-sided pregnancy is abnormal. In any literature to which the writer has had access, these opinions were not substantiated by published data. Two workers, Williams[7] and Gildow,[8] have gathered data on this subject which tend to give support to the view that right-sided pregnancy is more frequent.

For the past several years, the writer has been making observations pertaining to this matter in the course of making examinations for pregnancy. In making these examinations the uterine horn bearing the fetus and the ovary presenting the corpus luteum of pregnancy have been noted. Observations were made on cattle from eight to twelve weeks in gestation, by rectal palpation of the uterine horns, ovaries and middle uterine arteries, a procedure familiar to veterinarians. In almost all cases the observations made on an examination were confirmed by repeating the examination later.

In many of the cases reported, opportunity was afforded to determine the sex at the time of parturition. This made it possible to make observations relative to the theory (now largely discredited) that each ovary produces ova capable of developing into one sex only.

SUMMARY

Of the 704 cases of single pregnancy observed, 293 (42 per cent) were in the left uterine horn and 411 (58 per cent) in the right uterine horn.

In 560 of the above cases in which the sex is known, 292 (52 per cent) were males and 268 (48 per cent) were females. Of 292 males, 121 (42 per cent) were carried in the left horn and 171 (58 per cent) in the right horn. Of the 268 females, 107 (40 per cent) were carried in the left horn and 161 (60 per cent) in the right horn.

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TABLE I—*Summary of all single pregnancies in left or right uterine horn.*

GESTATION*	BREED										TOTALS		
	GUERNSEY		JERSEY		AYRSHIRE		BROWN SWISS		HOLSTEIN				
	L	R	L	R	L	R	L	R	L	R	L	R	BOTH
1	13	13	14	18	8	7	3	3	35	67	73	108	181
2	8	14	16	12	8	6	1	5	28	61	61	98	159
3	6	9	6	12	4	10	....	5	36	36	52	72	124
4	4	8	5	6	3	5	3	3	20	33	35	55	90
5	2	5	2	6	3	3	2	2	29	16	38	32	70
6	2	4	3	2	2	2	....	2	13	10	20	20	40
7	....	1	1	1	....	1	....	1	5	4	6	8	14
8	....	....	1	2	....	1	....	....	1	3	2	6	8
9	....	....	1	1	....	....	....	....	....	1	1	2	3
10	....	....	....	1	....	....	....	....	....	....	....	1	1
11	....	....	....	1	....	....	....	....	....	....	....	1	1
12	....	....	....	1	....	....	....	....	....	....	....	1	1
13	....	....	1	....	....	....	....	....	....	....	1	....	1
Unknown	....	....	1	2	....	....	....	2	3	3	4	7	11
Totals	35	54	51	65	28	35	9	23	170	234	293	411	704

*Number 1 means the first gestation or pregnancy, 2 the second, and so on. L = left uterine horn. R = right uterine horn.

TABLE II—*Summary of all single pregnancies in left or right horn, with sex of offspring when known.*

GESTATION	PREGNANT IN LEFT HORN			PREGNANT IN RIGHT HORN			TOTAL CASES
	CASES	MALE	FEMALE	CASES	MALE	FEMALE	
1	73	33	26	108	44	44	181
2	61	18	25	98	39	44	159
3	52	27	15	72	32	25	124
4	35	15	15	55	23	24	90
5	38	15	14	32	12	13	70
6	20	7	7	20	8	7	40
7	6	4	1	8	4	1	14
8	2	.....	1	6	2	1	8
9	1	1	.....	2	1	1	3
10	.....	.....	.....	1	1	.....	1
11	.....	.....	.....	1	1	.....	1
12	.....	.....	.....	1	.....	1	1
13	1	.....	1	.....	.....	.....	1
Unknown	4	1	2	7	4	.....	11
Totals	293*	121	107	411*	171	161	704

*It will be noted that the number of cases listed is larger than the total number of males and females reported. This is because, in many cases, the cattle were sold while pregnant. In other cases, parturition had not occurred at the time data were compiled.

TABLE III—*Further summary of data in table II, with breeds listed.*

BREED	PREGNANCY IN LEFT HORN			PREGNANCY IN RIGHT HORN			TOTAL CASES
	CASES	MALE	FEMALE	CASES	MALE	FEMALE	
Guernsey.....	35	18	8	54	21	25	89
Jersey.....	51	20	20	65	30	23	116
Ayrshire.....	28	12	11	35	14	16	63
Brown Swiss....	9	2	6	23	11	6	32
Holstein.....	170	69	62	234	95	91	404
Totals.....	293	121	107	411	171	161	704

There is evidence presented that either ovary produces ova which develop into individuals of either sex with approximately equal frequency. Of ova produced by the left ovary, 121 (53 per cent) developed into males and 107 (47 per cent) into females. Of ova produced by the right ovary, 171 (51 per cent) developed into males and 161 (49 per cent) into females. Seventy-seven cows were observed which are known to have produced both male and female offspring from the same ovary.

No authentic cases of migration were observed. In a few instances cows have been observed in which the corpus luteum of pregnancy disappeared from one ovary and another corpus luteum formed in the opposite ovary. In some of these cases ovulation may have occurred as symptoms of estrus were manifested.

Among 578 gestations which were completed, there were 17 of twin pregnancy. In seven cases where the calves were of opposite sexes, the fetal membranes were examined for anastomoses of blood circulation. In the four cases where an anastomosis was found, autopsy revealed a "free-martin" heifer, confirming the observations of Lillie.[9]

Lothe[10] and Clapp[11] report having observed an apparent relationship between the incidence of cystic ovary and twinning in cattle. In 18 cases of multiple pregnancy observed (one of triplets and 17 of twins), none of the cows had a history of cystic ovary before the gestation referred to or at any preceding gestation. In the same herds there were occasional cases of cystic ovary in other cows.

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Arthritis Specialists Exchange Views

The seventh annual meeting of the American Society for the Study of Arthritis was held at the Waldorf-Astoria, New York, December 12-14, 1935. The organization is made up of men who are specializing in the treatment of arthritis in the various sections of the United States and Canada. The program consisted of about a dozen papers on as many phases of arthritis, followed by discussions. Veterinarians will be interested in knowing that members of the medical profession are interested in learning more about arthritis in animals and to this end a paper entitled, "Diagnosis and Treatment of Arthritis in Horses," was presented by Drs. Cassius Way and George L. Hoffmann. According to reports, members of the Society were deeply impressed with the information contained in this paper, which was read by the senior author, Dr. Way.

Doctor Durrett Returns to Old Post

Dr. J. J. Durrett, of New York, has returned to his former post as chief of the Drug Division of the Food and Drug Administration, after an absence of a little more than four years. During that period, Dr. Durrett was engaged in commercial work and was associated with E. R. Squibb & Sons, manufacturing chemists. He originally entered the service of the Food and Drug Administration in 1928, and resigned in August, 1931. As chief of the Drug Division, Dr. Durrett will be in control of the work involved in the enforcement of those provisions of the Food and Drugs Act which apply to drugs, medicine and other preparations, including those sold for the treatment of diseases of animals.

Diner: "There's something wrong with these hot dogs."
Waiter: "Well, don't tell it to me. I'm only a waiter, not a veterinarian."

A CONSIDERATION OF URINARY CALCULI IN ANIMALS*

By C. E. DE CAMP, *Scarsdale, N. Y.*

Veterinarians are familiar with the seriousness of calculi in both large and small animals. The so-called "bladder-sand," commonly found, particularly in cats, is capable of causing much injury to the urinary tract. If present in sufficient quantity, it may so completely fill the urethra as to prevent the passage of urine.

The purpose of this paper is to present a brief consideration of the factors which bear directly on urinary calculi, which may aid veterinarians in more effectively handling this condition. No original work is reported in this paper, but a brief summary of the available literature on this subject will be made. Due to the extensive work which has been done in studying urinary tract abnormalities in the human field, I shall present data in part from this field, which may be applicable to veterinary work. I trust this will stimulate considerable discussion on the subject. It is hoped that by this means more interest in urinary diseases in animals will be created, and more valuable data from clinical observations will be accumulated.

The cause of urinary calculi is still unknown. Calculi are not a disease but a symptom. Permanent relief often follows after correction of the condition which produced the symptoms. It has been suggested that, at some time in the future, there will be found a fundamental, single cause, as yet undetermined, to account for stone formation, and that secondary factors will then enter to vary the actual chemical composition in individual cases. Many etiological factors enter into the formation of calculi. Those which are responsible in one individual may be inadequate in another.

Heredity, race, sex and age are all considered to be important as bearing on calculi in humans. Whole families are often found in which each member is subject to urinary calculi. It is interesting to note that the negro, in all parts of the world, is free from urinary calculi. No other race shows such a degree of immunity. Urinary calculi are more common in the male. As to age, one investigator reports his findings on 224 cases to show that those occurring in early life are due to continued dietetic errors, resulting in vesical calculi. Cases occurring in mid-life result mostly from inflammatory processes, with the stones

*Presented at the quarterly meeting of the Hudson Valley Veterinary Medical Association, Poughkeepsie, N. Y., November 13, 1935.

mostly in the kidneys. Most cases which appear during late years result from obstructions to the outflow from the bladder.

Geographical and climatic influences play a part, either directly or indirectly, on the cause of urinary calculi. Cattle raised in sections where green fodder is not easily available consume a diet which is necessarily dry and bulky. Such diets show an excess of lime and a scarcity of phosphates which are conducive to calculus formation. Stone is common in cattle in such localities, and the milk they produce is poor in vitamin A. In the United States, statistics recently collected show that the highest incidence of urinary calculi, in humans, is in southern California and in southern Florida.

The Role of Infection

Infection is decidedly the most serious complication of upper urinary tract lithiasis. The possibility of infection reaching the kidney and bladder can easily be understood by recalling the fact that these organs are constantly bathed in urine, a fluid which serves the important purpose of carrying away from the body large quantities of waste material and disease organisms. Infection causes a more rapid destruction of kidney tissue; its persistence leads to the inevitable destruction of kidney function. Its importance as a complication is recognized in the present-day standards of treatment which require, among other things, not only the removal of all stones, but the clearing up or eradication of all infections, local and focal.

Bacillary and coccal infections are the most commonly found in connection with urinary calculi. *Bacillus coli* is the most frequent of the former, and staphylococci of the latter. Chronic infections of the kidney often occur as complications of upper respiratory tract infections, septic wounds and various cutaneous infections. Urine often shows little or no evidence of infection and generally appears normal, but most investigators agree, however, that infection is present under these conditions. True bacterial calculi occur either composed entirely of dead bacteria, or a clump of bacteria may act as a nucleus for the precipitation of minerals in stone formation. Blood and fibrin also have been seen as nuclei of renal calculi.

An interesting study has been reported by Rosenau,[1] showing the relation of infection to calculi in dogs. The teeth of a group of normal dogs were artificially infected with cultures from teeth, tonsils and urine of human patients suffering with long standing cases of nephrolithiasis. Calculi, or lesions of the medulla of the kidney, were produced in 87 per cent of dogs

whose teeth, normal in the beginning of the study, were exposed to the action of the organisms mentioned. These calculi or lesions were similar in physical properties and chemical composition to those present in the urinary calculi in the men from whom the infecting organisms were obtained.

An additional etiological factor is the crystalline matter which is held in solution by true colloids normally existing in the urine. These colloids have a direct effect on the highly insoluble urinary substances which are of a protective nature. When these colloids are altered, agglutination and precipitation of the urinary substances take place. Change in the amounts of the colloids or crystalline substances upsets this balance and precipitation results, with the materials thrown down being ready for other factors to utilize in the formation of a stone. Colon bacilli and other organisms cause precipitation of the normal colloids of the urine. Staphylococci in the urine split urea, causing an increase of ammonia, resulting in an alkaline urine which bears directly on the formation of calculi. It can therefore be readily assumed that infection is a foremost cause of this urinary tract abnormality.

METABOLISM AND ELIMINATION

Faulty metabolism, diets unsuited to the individual, and improper elimination of wastes are all important contributing factors. Vitamin A and D deficiencies are now known to contribute in an important way to this disease. The effect results in keratinization of the epithelium of mucous membranes and pelvis of the kidney. These tissue changes produce nuclei for the deposition of minerals precipitated from the urine, and also open avenues for the entrance and development of pathogenic organisms.

It has been shown that for greatest effectiveness in preventing and clearing up urinary calculi, vitamin A in the diet should be of animal origin. Lack of sufficient phosphates to combine with the calcium present in diets increases the incidence of urinary calculi.

McCarrison[2] states that there are positive and negative factors in the relationship of dietetic errors to stone formation. The positive factors include an excess of calcium and an unknown agent in whole wheat grains. The negative factor may be a deficiency of vitamin A derived from animal sources or a deficiency of phosphates in relation to the amount of calcium in the diet. These factors, bearing directly on urinary calculi formation in humans, undoubtedly are of equal importance in animals.

Long continued restriction of fluids, resulting in an increase in the specific gravity of urine, may play a part. Renal calculi frequently follow fractures of the spine and pelvis. It is believed that fragility of the bones which accompanies nutritional diseases may so upset calcium metabolism as to make renal calculosis fairly common. Of equal importance is the fact that the urine remains free from bacterial and cellular elements in normal subjects, whereas these disease products are present in abnormalities of the urinary tract.

Irregular functioning of the parathyroid glands is considered to be a factor in the formation of urinary calculi. Their chief function is the regulation of calcium and phosphorus metabolism. When metabolism of a certain mineral element is out of balance, it usually disturbs metabolism of other minerals in the body.

Any condition which influences the reaction of the urine is important as a contributing cause. This is also important in treatment by the use of a ketogenic diet, as will be explained later. Phosphates are always deposited in alkaline urines, although all alkaline urines do not form calculi. Clinical experience shows, however, that an alkaline urine associated with such infections as *Micrococcus urease* often produces ammoniacal fermentations, and is commonly seen with bladder inflammations connected with calculus formation (Milks[3]).

Symptoms

Symptoms of the presence of urinary calculi are well known to veterinarians. It should be remembered, however, that vesical calculi, particularly those which are smooth, can remain in the bladder for a considerable length of time without producing symptoms. Often the first signs are frequent attempts to urinate, with very little success. Sudden checking of the voided stream may be the first symptom. If cystitis is present, blood and pus usually are found in the urine. Abdominal distension may be the first symptom noted, and will need to be differentiated from pyometra, and from large quantities of gas or fluid in the stomach or intestines.

Treatment, therefore, consists in a large measure of directly correcting the existing causative factors. A thorough physical examination of the patient should be given and urine analysis made, which usually produces extensive data on the cause of the malady. All sources of infection, both focal and general, should be eliminated. Special consideration should be given to diet, both as to its proper digestion and disposal, and as to its mineral and vitamin content. Adequate liquid intake must be main-

tained to prevent the urine from becoming concentrated and its pH thereby influenced. It is desirable, however, to keep liquid intake at a minimum whenever medication for the urinary tract is given orally. This prevents the medication from becoming too diluted and its therapeutic action made less effective when it reaches the site of infection.

The urinary tract being constantly bathed in urine, this fluid may be rendered antiseptic by the instillation of drugs and, under certain circumstances, by diet. For combating infection in the urinary tract, the use of azamine has produced results showing its efficient bactericidal action (Hoefle[4]). Administered orally, this medicament is eliminated chiefly through the urinary tract, exerting an effective germ-destroying action, particularly against *B. coli* and most pathogenic cocci. The deep penetration of azamine carries its desirable effect into the various parts of the organs it contacts (Spaulding[5]). Azamine is well tolerated, with a negligible minimum of irritation and cumulative action. In most cases it is equally active in either acid or alkaline urines. The importance of this will be appreciated when it is remembered that most urinary antiseptics require a certain pH of the urine in order to produce the desired effect.

KETOGENIC DIET

The use of a ketogenic diet in the treatment of urinary tract infection due to members of the *B. coli* group in humans has proven helpful in many cases. We refer to it here in the hope that such a diet may be utilized for the same purpose in animal urinary infections.

The value of the ketogenic diet is based on the fact that growth of members of the *B. coli* group is inhibited in urine which is markedly acid. A ketogenic diet produces such a urine and also throws off large numbers of the following ketone bodies: beta-oxybutyric acid, aceto-acetic acid and acetone. These ketone bodies also check the growth of *B. coli* organisms. The first named ketone body is the most important and must be present in 0.5 per cent or more, and the urine must carry a pH of 5.5 or less. Fatty acids of metabolism are ketogenic factors.

Ketosis, the condition produced in the urine as the result of feeding a ketogenic diet, is not synonymous for acidosis. Ketosis is an abnormal production of the three ketone bodies in the urine: beta-oxybutyric acid, aceto-acetic acid and acetone. Acidosis is an increase in the hydrogen-ion concentration of the blood plasma, or a diminution of the bicarbonates of the blood.

Glucose is antiketogenic. In a ketogenic diet, starch must be at a minimum as protein and fat are both, to a degree, glucose formers. A standard ketogenic diet for an adult human consists of a large amount of fat, an adequate amount of protein, and a minimal amount of carbohydrate. Tomatoes every other day are a suitable source of vitamin C to such a diet. Approximations of the substances constituting a ketogenic diet for an adult human are in the ratio of 1 gram of carbohydrates, 4 grams of proteins, and 20 grams of fats. Ketosis develops because the available glucose is inadequate to oxidize the fats completely, with the desirable ketone bodies being thrown off in the urine.

The ketogenic diet is given to humans at weekly intervals. It should produce ketosis in three to five days. If it does not, the diet is suspended for a week and then repeated every other week. During its suspension ammonium chloride or ammonium nitrate in enteric-coated pills is given, 15 to 30 grains daily. The patient must be closely supervised for any abnormal reactions which may occur as the result of this diet.

It is possible that this information from the human field can be utilized advantageously by veterinarians. It can easily be determined whether suspected urine contains a pH of 5.5 or less, as necessary to inhibit *B. coli*. This is done by adding one drop of chlor-phenol-red to 20 drops of the urine. If the color of the urine does not change, it shows that the pH is 5.5 or less, or the desired acidity is present. If the urine becomes pink or red, following the addition of this indicator, the acidity is insufficient or above 5.5.

Test for Ketone Bodies

A simple test for ketone bodies in the urine is: To 10 cc of suspected urine, add an equal volume of 10 per cent aqueous solution of ferric chloride. The development of a Bordeaux-red color indicates that ketone bodies are present in 0.5 per cent or more, the desired amount. It should be remembered that a moderate amount of salicylates in the urine will give a false reaction. By utilizing these simple tests, it is possible that the veterinarian can employ this valuable diagnostic information in treating urinary infections in animals due to members of the *B. coli* group. Inasmuch as this group of organisms is present as a causative factor in many intestinal tract infections, it can readily be understood how it can be so prevalent in urinary tract abnormalities.

Helmholz[6] summarizes as follows: "The ketogenic diet is a useful method for treating urinary infections resistant to alkalinization, diuresis and urinary antiseptics. Nephrolithiasis and

impaired renal function are two serious handicaps to the success of this form of treatment." The occurrence of new infections need also to be considered as being a possible cause in those cases where this diet has not proven to be of value. The use of a ketogenic diet, perhaps modified from its form in humans, may be helpful to the veterinarian.

An increased alkali content of the urine is useful in dissolving uratic calculi and cystine calculi, while acidification of the urine is indicated in eliminating calcium oxalate and calcium and ammonium magnesium carbonate and phosphate calculi. To acidify urine, a 10 per cent aqueous solution of concentrated nitrohydrochloric acid is suitable, as well as ammonium chloride or ammonium nitrate in suitable doses. To alkalize urine, magnesia, potassium citrate or acetate, or sodium bicarbonate is used. Proper acidification or alkalinization of the urine, as indicated for the particular case, can then be carried out for more effective results in eliminating the calculi present.

Deep anesthesia often permits sufficient urine and calculi to be passed to make catheterization unnecessary. Anesthesia is desirable also where a catheter is about to be passed, as it relaxes all structures, making way for easier instrumentation. Lavage, which should be carried out whenever possible, can best be conducted in the animal under anesthesia. For lavage, azamine in ½ per cent aqueous solution, or a 1 to 5 per cent aqueous solution of phosphoric acid is suitable.

More routine study of urinary calculi of animals will probably expand our knowledge of urinary tract abnormalities. An example of this is shown in the recent report by Morris and his associates,[7] of finding a cystine calculus in a dog, which, with other symptoms, showed the case to be cystinuria. This condition, to be sure, is not common, but it is an example of the vast amount of valuable information for veterinarians which may be produced by more detailed routine examination of urine and urinary calculi. Undoubtedly every veterinarian has access to a laboratory which can examine urinary excreta to determine their physical and chemical structure.

In closing, permit me to leave this thought for your consideration:—that more detailed use of diagnostic measures and methods of treatment in urinary infections, as developed in the field of human medicine, and applying this information as much as possible to urinary tract infections encountered by the veterinarian in his daily practice, will undoubtedly result in more information on urinary infections being obtained for the veterinarian. This, of course, will result in more effective methods of

treatment and will enable the practitioner to render greater service to his patients and clients.

Summary

1. The cause of calculi is unknown, and varies in different individuals.

2. Infection is a most important complication in calculus formation. Its removal is necessary for prompt and complete recovery.

3. Azamine is an efficient bactericide in urinary tract diseases.

4. Faulty metabolism and long continued restriction of fluids are important contributing factors.

5. The use of a ketogenic diet, modified to suit animal needs, may be valuable to the veterinarian in treating urinary infections caused by members of the *Bacillus coli* group.

6. Adequate amounts of vitamins A and D in the diet are necessary in the relief and prevention of calculus formation. This vitamin A should be of animal origin.

7. More attention given by veterinarians to examinations of urine and calculi will undoubtedly produce much valuable clinical data. More effective treatment can then be carried out.

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Printed in Ten Languages

A copy of the latest issue of the *Bloodless Phlebotomist* has been mailed to every veterinarian in the world with a known address, except in the countries of Russia, Latvia and Bulgaria. This little journal is published by the Denver Chemical Manufacturing Company, of New York, and contains much interesting information. It took 1,450,500 copies to send one to each physician and veterinarian on the mailing list of the publisher. It is printed in ten languages. If you did not receive your copy, write to the Denver Chemical Manufacturing Company, New York.

SKIN LESIONS IN CATTLE*

By W. M. PENDERGAST, *Syracuse, N. Y.*

Skin lesions in cattle have assumed a rather important significance in the tuberculin testing of cattle. This condition is rather widespread, although some localities seem to have more of it than others.

The lesions usually are found on the front and hind legs and, as a rule, below the knees and hocks. They are sometimes found in the region of the shoulder. The lesions may be quite extensive or there may be only one or two small nodules under the skin. Many of these nodules will show abrasions of the skin over the nodules, while others may show a discharge. On being incised, the nodules usually show a thick, yellow pus.

I have held several hundred postmortems on reactors showing skin lesions and in more than 95 per cent of these cases, I have failed to find internal lesions of tuberculosis.

Out of several hundred cases of skin lesions, I have observed probably less than five cases that showed tuberculous lesions of the prescapular lymph-gland, associated with skin lesions in the region of the shoulder.

The evidence seems to point to the fact that these skin lesions are caused by infection through the broken skin. This view is supported by the fact that most of the lesions are found on the lower extremities which are more subject to injury. I mention one case that tends to bear out this opinion:

An owner had ten reactors in a herd that had been accredited a year previously. I held the postmortem on these reactors and the owner, being somewhat disturbed, attended. The results of the postmortem were as follows: Four no-visible-lesion cases and six showing rather extensive lesions on the hind legs below the hock. The owner explained that he had a dog that would often grab the animals by the hind legs, and that was where the lesions had developed.

*Presented at the 26th semi-annual meeting of the Central New York Veterinary Medical Association, Syracuse, N. Y., November 9, 1935.

The peculiar fact about skin lesions is that we see many of them in accredited herds that have been through several tests. In the past 15 years, I have held postmortems on several thousand head of cattle. During that time I have observed very few cases of skin tuberculosis in animals that have reacted on the initial test. A great many of the animals that show nodules on the legs will show more or less reaction to the intradermal test. As a rule, the reaction is not typical. The swelling is apt to be more or less soft and diffused, although some will show a rather marked reaction. Animals in clean or accredited herds that show suspicious reactions should be carefully examined in the region of the legs and shoulders, for sometimes the skin lesions are very small.

In the relisting of clean or accredited herds, it is my opinion that the veterinarian should show considerable judgment before condemning animals, showing suspicious reactions, that are also affected with skin lesions.

MUMMIFIED TWINS IN A COW*

By W. M. PENDERGAST, *Syracuse, N. Y.*

Subject: A purebred Holstein cow about four years old.

The owner asked me to examine this cow, stating that she had been bred over eight months previously and had shown no sign of being in calf or having come in estrum. Upon examining the animal through the rectum, I felt a small, rather hard object, about six or eight inches long, in the uterus. I told the owner that I believed it was a mummified fetus.

Treatment: In treating the cow, I grasped the cervix of the uterus with forceps, and although the cervix was sealed, I gradually inserted a small metal catheter through the cervix into the uterus. I injected about eight ounces of a 2 per cent Lugol's solution, but was unable to get any return flow. The next morning the owner found a pair of mummified twins, about six inches long, in the drop behind the cow.

About one month later, I treated this cow as follows: I injected 2 per cent Lugol's solution into the uterus, and pressed it out by massaging the organ. I also swabbed the cervix with full strength Lugol's solution. This animal was bred a few days later, became pregnant and has had four healthy calves since that time.

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*Presented at the 26th semi-annual meeting of the Central New York Veterinary Medical Association, Syracuse, N. Y., November 9, 1935.

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MORTALITY IN YOUNG TURKEYS ASSOCIATED WITH TRICHOMONIASIS*

By A. G. GIERKE, *Sacramento, Calif.*
State Department of Agriculture
and W. R. HINSHAW, *Davis, Calif.*
University of California

The purpose of this report is to record observations made during the past three years on outbreaks of enteritis in young turkeys, characterized by the presence of countless numbers of flagellates of the genus Trichomonas in the lower intestines and ceca as the only suggestion of a causative agent.

History: For years, turkey-growers in California have experienced severe losses from undiagnosed causes among poults under eight weeks of age. In many of these outbreaks trichomonads were observed, but because of the general opinion tha they were normal inhabitants of the intestines, diagnosticians did not consider them as a possible factor in causing the mortality. In 1933, increased numbers of cases, with typical clinical and pathological characterizations, were received at the various diagnostic laboratories. In 1934 and 1935, outbreaks have been epizoötic in nature in several turkey-growing sections of the state. Poults from one to eight weeks of age suffered most. The average mortality was in the neighborhood of 50 per cent of the entire affected brood. Many growers lost 75 per cent of their turkeys during the past brooding season.

Symptoms: Listlessness, sagging of the wings, a general unkempt appearance and watery, foamy droppings are the principal symptoms. Cecal discharges vary with the feeding methods, but are usually yellowish brown in color, foamy and semi-liquid. No bloody diarrhea has been observed. Enormous numbers of trichomonads can be seen when examining a drop of the foamy liquid droppings with a microscope.

The course is rapid, usually a matter of hours, although the sick poults may linger two or three days. Just before death, they rest on their haunches with their heads lying on or under a wing. Slight convulsions usually precede death.

Autopsy findings: The crop is nearly always empty. The general condition of the poult indicates that it had not eaten for some time, before marked symptoms appeared. When the poult lives for a few days it rapidly becomes emaciated. Gross internal

*Received for publication, November 21, 1935.

lesions are confined to the intestinal tract, although the gall-bladder is constantly enlarged.

The entire intestine lacks tone, appears thin-walled, especially posteriorly, but shows little indications of inflammation. Occasionally, sections of the intestine below the duodenum are bulbous and slightly congested. The contents are liquid or semi-liquid and contain many gas bubbles. The cecal contents may be liquid or semi-liquid and contain considerable gas.

Microscopic findings: Myriads of trichomonads can be demonstrated in the contents of the lower intestine and ceca. Systematic examination of scrapings of the entire digestive tract from the mouth to the anus have been made on poults from several

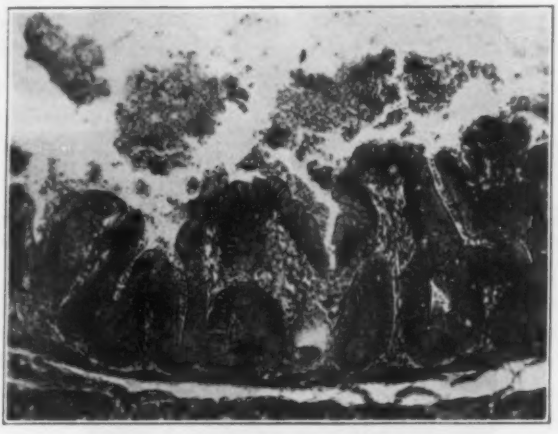

FIG. 1. Section of cecum showing masses of trichomonads (x 100).

outbreaks and the parasites in all instances have been confined to the lower two-thirds of the intestine. If flagellates are found in the upper third of the intestine they are usually confined to the bulbous inflamed areas. They have not been found in the blood-stream or other tissues of the body, by examination of fresh materials.

In many cases an abnormal number of spiral forms of bacteria were found in the cecal contents. Whether these have any relation to the disease is not known at present.

Coccidiosis and crop mycosis complicated some of the outbreaks studied and in these cases there was a somewhat different pathological picture.

Histopathology: Sections from the heavily infested ceca give no indication of how such severe reactions are produced. The epithelium is entire, showing neither desquamation nor proliferation, and although the crypts are filled with trichomonads, none are found in the epithelial cells or in any of the underlying structures.

Bacteriological studies consisted of plating of heart-blood, liver tissue, and in case of dead birds, bone-marrow, on meat extract, cooked blood-agar and an infusion medium made from turkey meat and liver. All these studies have yielded negative results. A limited number of anaerobic studies have also given negative results.

FIG. 2. Section of cecum. Crypts filled with trichomonads. Epithelium intact (x 420).

Blackhead lesions have not been associated with any of the outbreaks studied and *Histomonas meleagridis* has not been recognized in the microscopic preparations.

Transmission studies: The life history of the flagellate or flagellates associated with these losses is being studied by Miss Aileen Bonestell, of the Department of Zoölogy, University of California. The studies completed to date indicate a similarity of the organism found in the California outbreaks to the one found associated with blackhead by Hadley[1] and called by him

Trichomonas meleagridis. It differs from the one described by Volkmar[2] in that it has four flagella and localizes in the cecum and lower intestine rather than in the upper digestive tract. It also differs from Weinzirl's[3] species in that the *T. pullorum* described by him has three flagella.

The flagellate grows abundantly on Locke's egg-albumen, but soon dies out. It grows more slowly on Locke's egg-blood medium but remains alive for a longer period.

In preliminary transmission studies, five-week-old poults have been successfully infected both by feeding and contact methods, but the clinical picture has not been the same as in field cases. Symptoms have been either absent or mild, and the mortality has been nil. Two turkeys infected at five weeks of age were still shedding trichomonads in large numbers one year later. Two kittens given material orally and rectally fresh from an infected bird failed to establish the parasite or to produce symptoms.

Epidemiology: Field studies have shown that management plays an important rôle. The disease has usually started in a community on a ranch where some contributing factor such as low fertility, poor hatchability or insanitation existed.

Invariably adult birds have been on the premises in these initial outbreaks. In at least two outbreaks, adult birds were shedding large numbers of trichomonads, although appearing normal. In one instance seven turkey hens used to brood poults were examined. Feces removed from the cloaca of one were found infected. In another case, one out of four females purchased from an infected ranch had large numbers of the parasites in the lower intestine when killed for market. The fact that three turkey-growers experienced losses in 1934 and 1935 is further evidence of possible adult carriers. In all of these, trichomonads were present in large numbers, and the pathological pictures for each year were identical. On three ranches where losses occurred for the first time in 1935, adult stock had been purchased from growers that had outbreaks in 1934.

This condition has existed in every community that has been under the writers' observation, and in most instances the disease has spread in true contagious form, probably helped by the traffic from infected to non-infected farms. A survey of ranches in infected areas that have had no losses suggests two factors that may account for their success. One is complete isolation of growing from adult stock and the other is better management.

Added evidence that trichomonads play an important part is that only in those outbreaks having the clinical picture, and char-

acterized by the pathological findings reported, have they been found in any number. Since starting these observations, a careful search has been made for trichomonads in all specimens brought to the laboratory for diagnosis.

Baby ducks and baby chicks were found associating with diseased turkeys without apparent ill effects. On another ranch ten-day-old chicks cared for by the same person that looked after the turkeys showed no symptoms, and a few examined were negative for the parasite. Several other ranches reported chicks on the same premises without losses.

Control and treatment: It is obvious that until the exact cause of the losses is determined, it will be impossible to arrive at a satisfactory control program. Due to erratic practices of the owners, who are largely influenced by unskilled feed-service men and remedy salesmen who flood the stricken areas during such outbreaks, it has been difficult to obtain reliable data on treatment. That which has given the most promise is a restricted, easily digested diet, together with a 1:2,000 dilution of copper sulfate as a substitute for all drinking water for a few days. Sanitation, adequate warmth during cold nights, and shade during the hot days, have aided in reducing mortality. There is little doubt but that more losses have occurred by gross mismanagement and the use of quack remedies than from the disease itself.

Summary

An enteritis in young turkeys characterized clinically by watery, foamy diarrhea, a rapid course and by a heavy mortality, has been associated with marked trichomonad infection of the lower intestine and ceca. Clinical and epizoölogical data furnish evidence that these flagellates, the species of which is still undetermined, may play an important rôle in producing the disease. Preliminary transmission experiments were successful in establishing the parasites in the intestines of normal turkeys but the clinical picture of the disease was not reproduced by artificial inoculation.

Field studies indicate that insanitation, feeding methods and faulty management may be influencing factors. Adult carriers appear important in the transmission of the parasites to the young birds.

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BOVINE ENCEPHALOMYELITIS IN MEXICO*

By JAVIER ESCALONA *and* FERNANDO CAMARGO
Mexico, D. F., Mexico

Observations on bovine encephalomyelitis in Mexico have been made over the last 30 years, but no research work was done until 1928. The common name for this disease among stockmen and farmers is "Derriengue" or "Tronchadeo" which means loin disease. It attacks horses, mules, burros and hogs and has been observed in wild animals such as deer and coyotes. It is more noticeable among cattle than any other species because of their economic importance, and is observed only on the west coast of Mexico. It has been noticed mostly in altitudes of between 800

FIG. 1. Cow down with encephalomyelitis.

and 1,200 meters. The first report of this disease was from the state of Colima. After this a spread of the disease southward was noticed, with reports of the condition in Costa Rica. At the same time, it spread northward but no report of this condition has come from the state of Sonora, which probably might be due to the fact that this state is tick-free.

Encephalomyelitis is the scientific term for this disease. The principal symptoms are excitability, tendency to attack, blindness, staggering gait, and finally the animal becomes paralyzed and sinks to the ground. This paralysis is mostly of the hind quarters, which is total, but the animals also show tetanic spasms

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of various groups of muscles of the neck and jaws. The owner usually notices a gritting of the teeth and a total loss of deglutition. The neck is usually drawn to one side or the other and remains in that position until the animal dies. The duration of the disease is from one to eight days. The mortality is practically 100 per cent. In all the cases observed, only one, in the state of Sinaloa, made a partial recovery after several months. This animal had an atrophy of the muscles of the posterior part of the body, causing a staggering gait and in grazing it fell down a creek bank and died. The loss from this disease is thought to be many millions of pesos a year.

In our experiments we used guinea pigs, rabbits, mice, cows and pigeons. In these we made subdural injections of diseased brain tissue according to the Shahan and Schoening method, as

FIG. 2. Another case of bovine encephalomyelitis.

used for equine encephalomyelitis. We found five strains of virus in five different states and made a fixed virus for each one. This fixation was made in guinea pigs in six days. The period is ten days in rabbits. In using the brain tissue we found intraocular injections more rapid than intramuscular and we also proved the saliva to be infectious by using intraocular, intramuscular and subcutaneous injections. We also produced an infection by scarification of the nasal membranes with infectious saliva. The bovine virus differs from the equine in that it does not affect pigeons.

All kinds of drugs were used in trying to treat this disease but the results were negative. We are now experimenting with a vaccine similar to that which is made for the equine disease. However, we have not enough data on which to base a positive

conclusion. We think, though, that the results from this will be very satisfactory. The best results obtained were where the owner was advised to dip against ticks. In every case, where this advice was carried out, the outbreak of the disease was stopped. Hence our conclusions that the tick is a carrier of this disease. It also proves that this is an enzoötic type of disease rather than one of an epizoötic nature.

For controlling this sickness we recommend, besides vaccination, cleaning the land of ticks, and enforcing all sanitary methods recommended by the Mexican government. These are segregation, destroying the sick, burning carcasses, disinfection, and quarantine of infected territory.

FOREIGN BODIES IN THE GASTRO-INTESTINAL TRACT OF THE DOG*

By J. GILBERT HORNING, *Houston, Texas*

The subject, a Scottish Terrier, two years old, weighing about 20 pounds, was presented on July 15, with a history of playing with a rubber bone. A few days before, the bone had been chewed to pieces and the dog had swallowed some of it, then vomited it up, but there probably was a piece left in the stomach, because the bone could not be pieced together completely.

When the dog arrived at the hospital, he was still vomiting. He was fed bread and cereals, and then given emetics. All of the food was vomited but there was no evidence of any rubber bone. High enemas were given the dog, but with no result. The foreign body still remained in the stomach, x-ray examination showing it to be as big as ever (fig. 1). Gastroscopy was performed, the foreign body removed, and the dog discharged from the hospital two days afterward, normal in every respect. From the size of the object it seemed as though this should have been vomited after the administration of emetics, high enemas, and the constant effort of the dog itself to expel the intruder, but such was not the case. Hence the reason for this case report being presented. Here was a case that should have responded to the treatment as outlined in the first part but it did not. X-rays followed by gastroscopy, in my opinion, saved this dog's life.

I recall a case similar in nature that was brought into our clinic some years back. This dog had a history of playing with a rubber bone. However, the dog expelled part of it by vomiting and some more after it was given emetics. At intervals of sev-

*Received for publication, October 30, 1935.

eral months, this dog was brought back to our clinic to be treated for nausea. No x-ray examinations were ever made and the dog was treated with the usual procedure for such conditions of the gastro-intestinal tract. He responded to this treatment, and was brought back, a few months later, showing the same symptoms. Nine months afterward, when brought back for treatment with nausea and gastric distress, he did not respond but died within 48 hours after entering the hospital. We asked permission to hold a postmortem and found a piece of rubber bone stopping at the ileo-cecal valve. This piece of rubber bone had been in the intestine so long that, although when first swallowed it was solid, through the effects of the gastric and intestinal juices, it had become soft and porous.

Hence I recommend that, in any case where there has been a possibility of foreign bodies entering the stomach, the client be advised that it would be safer to have an x-ray examination

FIG. 1. X-ray photograph showing foreign body (portion of rubber bone) in stomach of dog.

to prove conclusively that there is nothing further left in the stomach and, when shooting the picture, shoot one of the whole body.

The x-ray machine used for taking this picture was a Little Giant. It is a small, complete, portable unit that so far has proved satisfactory in our work.

Visitors at the Journal Office

The various meetings held at Chicago during the first week of December brought several hundred veterinarians to the Windy City. A number of these took a few minutes off to visit the A. V. M. A. office. The first visitor during the week was Dr. R. A. Runnels, of Iowa State College, who spent the past summer in Europe. Dr. Edward Records, of Reno, Nevada, president of the U. S. Live Stock Sanitary Association, was the next caller. He was followed by Drs. H. C. H. Kernkamp and B. S. Pomeroy, of Saint Paul, Minn. Dr. Kernkamp was in Chicago to attend his first meeting as a member of the A. V. M. A. Executive Board. Then came Dr. J. C. Flynn, of Kansas City, Mo., for his first visit to the office as President of the A. V. M. A.

Drs. R. R. Dykstra and H. F. Lienhardt, of Kansas State College, dropped in for a brief inspection of the office, as did Dr. Wm. Moore, of Raleigh, N. Car., and Dr. M. Jacob, of Knoxville, Tenn. Dr. Moore is a member of the A. V. M. A. Executive Board, and Dr. Jacob is A. V. M. A. Treasurer. Dr. Adolph Eichhorn, of Pearl River, N. Y., called to discuss some matters in connection with the International Veterinary Congress, and Dr. R. P. Marsteller, of College Station, Texas, dropped in to talk over several questions relating to veterinary education.

Robert M. Chapin Succeeds Doctor Dorset

The U. S. Department of Agriculture recently announced the appointment of Robert M. Chapin as chief of the Biochemic Division of the Bureau of Animal Industry, succeeding the late Dr. Marion Dorset. Mr. Chapin is a native of Massachusetts, and a graduate of Amherst College. He also took postgraduate work in chemistry in that institution, and served as an instructor in chemistry until 1907, when he entered the employ of the Bureau of Animal Industry. Since the death of Dr. Dorset, in July last, Mr. Chapin has served as acting chief of the Biochemic Division.

ON NATURAL ANTIBODIES IN THE RABBIT AND HEREDITARY RESISTANCE TO INFECTIONS OF BRUCELLA SUIS. M. R. Irwin and F. N. Bell. Jour. Inf. Dis., lvii (1935), 1, p. 74.

Low and high bactericidal action of citrated whole blood of rabbits was highly correlated with susceptibility and resistance to *Brucella suis*. Rabbit blood in these tests had a much greater bactericidal action against *Br. abortus* than against *Br. suis*. Further specificity of the bactericidins was shown by the independent action towards *Br. suis* and *Salmonella enteritidis*.

THE SURVIVAL OF THE VIRUS OF LARYNGOTRACHEITIS IN THE BURSA OF FABRICIUS AND CLOACA OF CHICKENS AFTER "INTRABURSAL" INJECTION. J. R. Beach. Jour. Inf. Dis., lvii (1935), 2, p. 133.

Active laryngotracheitis virus was not detected in the bursa of Fabricius of young chickens later than the seventh day following injection of virulent virus into the bursa. This time interval corresponds to that during which the visible inflammatory reaction of the cloacal mucous membrane to the virus was present in the chickens. These experiments suggest that in about one week after vaccination against laryngotracheitis chickens cease to be virus carriers.

THE SURVIVAL OF TUBERCLE BACILLI SUBJECTED TO A VACUUM OF HIGH ORDER. Truman Squire Potter. Jour. Inf. Dis., lvii (1935), 2, p. 149.

Massed cultures of six avian strains of tubercle bacilli were sealed *in vacuo* in Pyrex tubes and stored in a dark room at room temperature. They were capable of inducing generalized tuberculosis when injected intravenously into pigeons, 5 and 8 months later. A single strain of tubercle bacilli similarly treated for 80 days remained viable when tested in a like manner. A single culture similarly treated but also placed under severe oxygen deprivation and stored at 37° C. in a dark room

for 17, 38 and 48 days also remained viable and capable of inducing disease in pigeons and rabbits. Oxygen deprival accompanied by maximal desiccation *in vacuo* at 35° C. in a dark room failed to render the tubercle bacilli incapable of inducing disease, 3 and 5 months later. There was no indication of the number of organisms which lost their viability or in the relative pathogenicity of the cultures following such treatment. The question is raised as to whether the organisms survive in their usual form or in some other form.

SPECIES OF CALLIPHORIDIAE CONCERNED IN THE PRODUCTION OF MYIASIS IN DOMESTIC ANIMALS, MENARD COUNTY, TEXAS. H. E. Parish and E. W. Laake. Jour. Parasitol., xxi (1935), 4, p. 264.

This study was undertaken to determine flies that are responsible for wound myiasis in Menard County, Texas. Sheep, goats, calves and grown cattle infested with maggots were placed in stalls; the larvae were trapped as they dropped from the wounds of the host. *Cochliomyia* spp., the screw worm flies, composed over 99 per cent of the total of all adults reared from larvae found in wounds during the summer and fall months. *Phormia regina* Meig, the black blowfly, constituted 67 per cent of all adults reared from larvae developed in wounds during the spring months of 1933. *Sarcophaga plinthopyga* Weid, a flesh fly, was found in very limited numbers in wounds of the animals used.

ON THE RESISTANCE OF THE CAT TO SUPERIMPOSED INFECTION WITH THE ASCARID, TOXOCARA CATI. Merritt P. Sarles and Norman R. Stall. Jour. Parasitol., xxi (1935), 4, p. 277.

Cats of different age groups and carrying natural infestations of *Toxocara cati*, were found to possess a uniformly high degree of resistance against attempts to superimpose infection with this ascarid. Two groups of factors seem to be involved in the resistance: (1) those limiting drastically the number of larvae reaching the small intestine and including failure of some eggs to hatch, loss of some eggs and larvae through vomiting and passage through feces and loss of some larvae during migration through the host's tissues; (2) those acting on ascarids which have migrated and returned to the small intestine, and including apparent prevention of growth or retardation of growth with shortened duration of infection and decreased egg production

of worms and expulsion by vomiting or passage in the feces of worms in all stages of development.

EFFECTS OF PREGNANCY URINE ADMINISTRATION TO FEMALE SWINE. E. P. Tschernozatonskaja. Endocrin., xix (1935), 4, p. 413.

Sterilized pregnancy urine was administered to female swine to determine if a depressing effect upon the sexual activity of these animals could be obtained and also to determine if such treatment would be feasible for use during the fattening period before marketing. Prolonged administration of large doses of pregnancy urine to 20 female swine, four to five months old, seemed to cause permanent inhibition of sex activity. Degeneration changes were found in the ovaries of a treated animal that was slaughtered. The ovaries were about twice the size of the ovaries of the control animals. The carcass weight, fat weight and depth of superficial fat were greater in the treated animals.

THE EFFECTS OF INJECTION OF RESIDUAL OVARIAN EXTRACTS. H. W. Marlow and Frederick Groetsema. Endocrin., xix (1935), 4, p. 415.

Purified extracts were made of sow's ovaries from which the follicular fluid and corpora lutea had been removed. The effects were studied in experiments involving 175 animals. When injected into 21-day-old female albino rats, the extract caused precocious sexual development. The males were not noticeably affected. The diameter of the uteri of the experiment animals were two to three times normal in seven to ten days. They did not remain in heat longer than the normal length of time. Pituitary bodies from donors that had received the purified extract until their vaginas were opened, were implanted, one daily, for ten days. At the end of this time the vaginas of the receivers were opened. Implants made from animals of the same age as the above animals, but receiving pure olive oil, had no effect on their receiver.

FURTHER OBSERVATIONS ON PRENATAL HOOKWORM INFECTION OF DOGS. A. O. Foster. Jour. Parasitol., xxi (1935), 4, p. 302.

The administration *per os* of single doses of infective larvae of *Ancylostoma caninum* to three pregnant bitches at intervals

of 5, 9 and 32 days, respectively, before parturition resulted in prenatal hookworm infestation in all but one of 21 puppies. Although the mothers were very resistant to hookworms, the puppies did not appear to be protected on this account. One litter of five pups was prenatally infected with the "cat strain" of *A. caninum* as the result of administering larvae to the mother two days before the birth of the puppies. The author suggests that the occurrence of prenatal infection with parasite worms may be of economic and public health importance.

INHERITANCE OF WRYTAIL IN JERSEY CATTLE. F. W. Atkeson and J. R. Warren. Jour. Hered., xxvi (1935), 8, p. 331.

Wrytail is the term applied when the base of the tail is set at an angle to the backbone. Four out of 15 calves in a single herd sired by one Jersey bull being wrytailed led to the study of this character. Three consecutive generations of normal sires were proved to be carriers of the factor for this character which is definitely inheritable. The character is widely disseminated through the Jersey breed and possibly other breeds of dairy cattle. Wrytailed animals of either sex should be discarded from the herd.

A LETHAL GENE IN JERSEY CATTLE. Carl Wipprecht and W. R. Horlacher. Jour. Hered., xxvi (1935), 9, p. 363.

An inherited lethal condition which is called "hairless" exists in the Jersey breed of cattle. This hairless lethal defect appears to be caused by a single gene which is a simple recessive. There is no definite time of expulsion of the hairless fetus by the various dams. The hairless defect appears to be closely associated with the condition known as wrytail. The latter is common but has received little attention. This hairless gene is not sex-linked as both male and female hairless calves have been born. Twenty matings of a heterozygous bull with daughters of bulls known to be heterozygous for the hairless gene were not sufficient to prove the heterozygosity of one bull.

Proofs of Our Weakness

But man has other enemies more formidable, against which he has not provided with such means of defense. These are the natural infirmities of infancy, old age, and illness of every kind, melancholy proofs of our weakness, of which the first two are common to all animals, and the last chiefly to man in a state of society.

—JEAN JACQUES ROUSSEAU, from, *"The Origin of Inequality."*

Regular Army

Major Nathan M. Neate, now sick at Walter Reed General Hospital, Army Medical Center, Washington, D. C., is relieved from further assignment and station at the San Francisco port of embarkation, Fort Mason, Calif., is assigned to station at Army Medical Center, Washington, D. C., and will report for duty accordingly.

1st Lt. Velmer W. McGinnis is relieved from further assignment and station at Fort Des Moines, Iowa, and will proceed to Fort Leavenworth, Kan., for duty.

1st Lt. Lloyd C. Tekse is relieved from further assignment and station at Fort Snelling, Minn., effective on or about November 20, 1935, will then proceed to Fort Francis E. Warren, Wyo., and report to the commanding general for duty.

Major Sawyer A. Grover is relieved from further assignment and duty at Fort Leavenworth, Kan., effective on or about December 1, 1935, and directed to proceed to Fort Sheridan, Ill., for duty.

The promotion of Lt. Col. Burton A. Seeley to the grade of colonel with rank from November 27, 1935, is announced.

Veterinary Reserve Corps

PROMOTIONS

To

Greissman, Louis	Major	Blauvelt Rd., Nanuet, N. Y.
Hamman, Fred Irwin	Capt.	P. O. Box 216, Loveland, Colo.
Bechtol, Lauren Leonhardt	1st Lt.	123 E. Butler St., Bryan, Ohio.
Bogaard, Kenneth	1st Lt.	864 A Ave., N. E., Cedar Rapids, Iowa.
Lange, Chester Jay	1st Lt.	72 Barrow St., New York, N. Y.
Smiley, Henry DeVore	1st Lt.	R.R. 2, Junction City, Kan.

NEW ACCEPTANCES

Martin, Earl Thompson	Capt.	532 Darlington, Tulsa, Okla.
Sullivan, Wm. Goodman	1st Lt.	2315 G St., South Omaha, Omaha, Neb.
Moats, Arthur Evans	2nd Lt.*	Waverly, Ohio.
Goldhaft, Tevis Mirsky	2nd Lt.*	E. Landis Ave., Vineland, N. J.
Versluis, Hendrick	2nd Lt.*	State Institute, Farmingdale, L. I., N. Y.

*Appointments were made before changes governing appointment in the Veterinary Section of the Officers' Reserve Corps became effective. New appointments are being tendered by the War Department in the grade of first lieutenant to all second lieutenants of the Veterinary Corps Reserve, coming within the provisions of these changes, as rapidly as possible.

Veterinary Corps Reserve

In order that the authorized grades of the Veterinary Corps Reserve may conform to those authorized for the Veterinary Corps of the Regular Army, the following changes, governing appointment in the Veterinary Section of the Officers' Reserve Corps, are announced by the War Department.

All future original appointments in the lowest grade of the Veterinary Corps Reserve will be made in the grade of first lieutenant.

All second lieutenants of the Veterinary Corps Reserve in a status of eligibility for assignment, active duty, and promotion in peace time, will be tendered new appointments in the grade of first lieutenant upon the submission of a satisfactory report of physical examination.

Veterinary Corps Reserve officers appointed first lieutenants will be credited with all their service as second lieutenants, Veterinary Corps Reserve, in computing the minimum time in grade for promotion to the grade of captain under the present regulations governing time in grade requirements for promotion in the Officers' Reserve Corps.

Second lieutenants of the Veterinary Corps Reserve now ineligible for assignment, active duty, and promotion in peace time will not be tendered appointments as first lieutenants, Veterinary Corps Reserve, unless they first meet the full requirements for restoration to a status with full eligibility now laid down by Army Regulations. If they do not meet these requirements, they will not be tendered reappointments at the expiration of their current five-year appointments. This is in conformity with current regulations. In case a second lieutenant, Veterinary Corps Reserve, now on the ineligible list, fully reëstablishes his status on the eligible list within the period of his current appointment, he will be tendered an appointment as first lieutenant, Veterinary Corps Reserve, upon submission of a satisfactory report of physical examination.

No change in status will be made in the case of former second lieutenants, Veterinary Corps Reserve, now in the Inactive Reserve (former Auxiliary Section, Officers' Reserve Corps).

Iowa and Montana Accredited

On December 1, the states of Iowa and Montana were placed in the so-called modified accredited area, by the U. S. Department of Agriculture. These two states bring the total up to 33, or over two-thirds of the total number of states. The accreditation of Iowa is peculiarly significant on account of the difficulties which delayed the tuberculosis eradication project in that state. Practically every hindrance was placed in the way of the veterinary officials in Iowa by the misguided stock-owners opposing the tuberculin test. A number of court actions became necessary before the work could be carried forward peacefully.

In Montana the degree of tuberculosis infection was generally low throughout the state, the principal problem involved being the testing of large numbers of range cattle which were not accustomed to being handled. However, the veterinarians assigned to the work in Montana slowly but surely overcame all obstacles on this account.

California Makes Important Change

At the recent session of the California Legislature, the position of Chief of the Division of Animal Industry of the Department of Agriculture was given a civil-service status. Previously the position had been filled by appointment by the Director of the Department of Agriculture, subject to the pleasure of the Governor. Taking the position out of politics and placing it

DR. C. U. DUCKWORTH

under civil service is expected to stabilize the administration of the office to a very considerable degree, in the opinion of California veterinarians. As far as is known, Wisconsin is the only other state that has placed the office of state veterinarian, or its equivalent, under the protection of civil service.

In addition to administering the control of infectious diseases of animals, the California Division of Animal Industry also includes the Dairy Service, Meat Inspection Service, Cattle Protection Service, Poultry Control Service, and the Dairy and Pathological laboratories. A combined personnel of 225, of whom 165

are veterinarians, is required to carry on the various activities of the Division.

Dr. C. U. Duckworth (Ind. '21) is Chief of the California Division of Animal Industry and is the first incumbent to enjoy a civil-service status. He rose from the ranks to the position he now occupies, having joined the Department of Agriculture as assistant field veterinarian in 1921. Dr. Duckworth advanced progressively to field veterinarian, market milk specialist, supervising market milk specialist, and Chief of the Bureau of Dairy Control. He resigned from the latter position to go with one of the large dairy companies. In 1933, he was appointed to his present positions as Chief of the Division of Animal Industry and Assistant Director of the Department of Agriculture.

New York Broadcasting Program

The New York State Veterinary Medical Society announces the following programs to be heard on veterinary subjects over WGY (General Electric Station), for the first six months of 1936.

January 9—"The Jones-Connelly Act and Your Dairy Herd," Dr. B. J. Cady, Bureau of Animal Industry, U. S. Department of Agriculture, Albany.

January 23—"Indigestion in the Horse," Dr. A. L. Smith, Mechanicville.

February 6—"The Next Lamb Crop" (Acetonemia or Pregnancy Disease of Ewes), Dr. C. E. Hayden, New York State Veterinary College, Ithaca.

February 20—"Team Work," Dr. James L. Shorey, Schenectady.

March 5—"Bang's Disease Control," Dr. R. R. Birch, New York State Veterinary College, Ithaca.

March 19—"Improving Milk Quality by Means of Frequent Udder Examination," Dr. J. H. Hewitt, Borden's Farm Products Company, Cortland.

April 2—"Blackleg Prevention," Dr. Chas. Linch, Department of Agriculture, Albany.

April 16—"Horse Sense About Horses," Dr. L. L. Parker, Catskill.

April 30—"Disinfectants—Antiseptics and Their Use on the Farm," Dr. D. W. Baker, New York State Veterinary College, Ithaca.

May 14—"Shoeing the Farm Horse," Prof. Henry Asmus, New York State Veterinary College, Ithaca.

May 28—"Your Pet Troubles" (Pets—Their Care and Management), Dr. A. J. Allott, Newburgh.

June 11—"Taking the Gamble Out of Breeding," Dr. S. A. Johnson, Kinderhook.

June 25—"Schenectady Plan for the Supervision of Meat and Milk," Dr. J. B. Garlick, Commissioner of Health, Schenectady.

These talks will be broadcast on alternate Thursdays at 12:45 p. m., Eastern Standard Time. Dr. John F. Miller, of the State Health Department, Albany, is chairman of the Radio Committee.

ORGANIZATION OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION, 1935-1936

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CENTRAL NEW YORK VETERINARY MEDICAL ASSOCIATION

The twenty-sixth semi-annual meeting of the Central New York Veterinary Medical Association was held at the Onondaga Hotel, Syracuse, November 9, 1935. Roll call showed 30 members present. Dr. Joseph Mosher presided.

Dr. A. H. Ide, of Lowville, gave a summary of the action that had been started against him for alleged malpractice. He read an article from his local newspaper which gave a full account of the plaintiff's side of the action. Dr. Ide then gave his side of the story and the case was discussed at length by Dr. J. C. Stevens and others. Dr. W. L. Clark then examined the minutes of the previous meetings of the Association and found where a motion prevailed to create a fund to be known as the Central New York Veterinary Medical Association Special Fund, to be used exclusively for defending such malpractice suits. It was found, however, that at a later date, the money in this fund had been ordered drawn from the bank and placed in the general fund. After much discussion, Dr. E. E. Dooling, on behalf of the Board of Censors, moved that the Association stand all the expense of the suit against Dr. Ide, and that the latter be allowed to select his own legal counsel. The Chair appointed a committee consisting of Drs. J. C. Stevens, J. B. Knapp and W. L. Clark to assist Dr. Ide when his case comes to trial.

Dr. W. M. Pendergast, of Syracuse, read two interesting case reports entitled, "Mummified Twins in a Cow" and "Skin Lesions in Cattle." (These reports are published in this issue of the JOURNAL.)

Dr. H. C. Stephenson, of the New York State Veterinary College at Cornell University, read a very interesting and instructive paper on small-animal practice, which was followed by a good discussion.

President Mosher extended a very hearty invitation for the organization to meet at his place at Genoa, in June, 1936. This invitation was accepted and the meeting adjourned.

At 6 o'clock the members gathered around the banquet table and partook of a splendid dinner. Quite a number of the ladies joined the gentlemen for the repast. Judge Frank Cregg was the speaker of the evening. He paid high tribute to the veterinary profession, in his talk, which was very much appreciated. He was extended a hearty vote of thanks.

W. B. SWITZER, *Secretary.*

MASSACHUSETTS VETERINARY ASSOCIATION

The regular monthly meeting of the Massachusetts Veterinary Association was held at the Hotel Westminster, Boston, on the evening of November 20, 1935. A good turn-out of members was present to hear Dr. D. M. Rioch, Assistant Professor of Anatomy at Harvard Medical School, talk on "Spinal Injuries in Dogs and Cats."

Dr. Rioch reviewed the anatomy and physiology of the spinal cord and gave many practical points on the diagnosis and prognosis of cord injuries. Among the many diagnostic facts brought out were that cord transection in the thoracic region results in paralysis of the rectum and bladder while injuries in the lumbar region leave the rectum and bladder with some tonus so that the urine will dribble. However, these reflexes may be altered by intercurrent infection. To diagnose complete transection, one must be sure that no impulses originating in the brain are reaching the hind legs or tail, for example, wagging of the tail on seeing his master or walking movements, coördinated with the front legs.

In order to make accurate prognosis, the x-ray is very helpful. In cases where there is some doubt about the extent of the cord injury, one should wait a week, and then, if there is evidence that the cord has not been completely severed, one may be hopeful for improvement for 18 months.

In response to queries, Dr. Rioch stated that rigidity of the fore limbs, so commonly seen soon after spinal injury in the dog, is due to bruising of the meninges and does not necessarily indicate cord damage. He also stated that he did not know the cause of paraplegia in Pekes and other long-bodied dogs, but thought that it might be due to toxemia, infection or trauma. The latter could be ruled out if, on lumbar puncture, there was no evidence of blood in the spinal fluid.

GERRY B. SCHNELLE, *Reporter.*

WESTERN NEW YORK VETERINARY MEDICAL ASSOCIATION

The twenty-second annual meeting of the Western New York Veterinary Medical Association was held in Buffalo, December 12, 1935.

The meeting opened at 2 o'clock, with clinics at the Erie County S. P. C. A. building. These consisted of a double entropion operation on a Chow, a case of a peculiar nervous disorder in a Scottish terrier presented for observation, two cases of posterior paralysis in dogs, a case of perineal hernia in a dog, and two cases for oöphorectomy.

Following the clinics, a business meeting was held. Officers were elected as follows: President, Dr. H. F. Wilder, Buffalo; vice-president, Dr. D. D. Ford, Niagara Falls; secretary-treasurer, Dr. F. F. Fehr, Buffalo (reëlected). Two new directors were elected to take the places of Drs. G. D. Stone and D. D. Ford. They are Drs. R. H. Arnett, Medina, and W. A. Dennis, Jamestown. Four new members were admitted: Drs. B. J. Stockler and R. H. Gaetz, Buffalo; B. J. Laughlin, Lockport, and P. T. Combs, Leroy.

After a banquet at Hotel Touraine, to which the ladies were invited, the evening was devoted to the following literary program:

A paper, "The Prevalence and Prevention of Myiasis Caused by the Grey-Flesh Fly (*Wohlfahrtia Vigil*)," by Dr. A. A. Kingscote, Ontario Veterinary College, Guelph, Ontario, Can.

An illustrated talk on some highlights from the experience of a laboratory diagnostician, by Dr. D. W. Baker, New York State Veterinary College, Ithaca.

A very interesting symposium on diseases of small animals, by Dr. R. H. Volgenau, Buffalo, followed by a discussion.

A very concise report of the A.V.M.A. meeting held at Oklahoma City, Okla., in August, by Dr. F. E. McClelland, Buffalo.

After some brief remarks by our Canadian visitors, Dr. W. J. Rumney, Hamilton; J. A. Campbell and H. S. C. MacDonald, Toronto, the meeting was adjourned.

F. F. FEHR, *Secretary.*

NEBRASKA STATE VETERINARY MEDICAL ASSOCIATION

The thirty-eighth annual meeting of the Nebraska State Veterinary Medical Association was held at Columbus, December 10-11, 1935. The attendance was slightly below average, due to

the fact that so many Nebraska veterinarians are employed temporarily by the U. S. Bureau of Animal Industry and the Nebraska Bureau of Animal Industry.

Dr. J. C. Flynn, of Kansas City, president of the American Veterinary Medical Association, headed the scientific program with a lecture covering small-animal practice. Dr. Flynn paid particular attention to canine distemper, as it is seen by the average practitioner.

Dr. H. E. Kingman, veterinarian for the Wyoming Hereford Ranch, at Cheyenne, Wyo., presented a blackboard lecture on functional sterility, which was highly instructive. On the second day of the meeting, Dr. Kingman again contributed to the program with a talk on cattle practice, with particular attention to mastitis.

Dr. L. Van Es, of the University of Nebraska, handled the subject of poultry diseases. Dr. F. M. Wilson, of Mechanicsville, Ia., presented the subject, "Equine Digestive Disturbances." The success of the annual clinic of the Eastern Iowa Veterinary Association, so well known to all practitioners in the Middle West, is due largely to the efforts of Dr. Wilson each year.

Dr. W. J. Moslander, of Grand Island, read a paper on "Equine Influenza," covering the work of himself and Dr. A. A. Anderson, in handling many hundreds of cases annually in the second largest horse market in the world. It is needless to say that this was one of the most valuable contributions to the program. A résumé of their work has been published in Spanish for the benefit of veterinarians in South America.

Dr. R. R. Dykstra, of Kansas State College, conducted a round-table discussion on equine practice. Dr. L. J. Boulier, of Cozad, contributed a number of interesting case reports dealing with swine erysipelas. Dr. Boulier probably has had as much experience with this disease as any practitioner in Nebraska. Many interesting points were brought out in his discussion. Dr. A. T. Kinsley, of Kansas City, Mo., then conducted a round-table discussion on swine diseases.

An invitation was extended to all professional men and women, including city and school officials, to view a special showing of a motion-picture film depicting rabies in animals and man. Approximately 75 persons accepted this invitation.

The banquet on Wednesday evening drew a crowd of 150. Local arrangements were in charge of Dr. and Mrs. E. J. Jelden, Dr. and Mrs. C. M. Curl and Dr. H. C. Groeteke. A turkey dinner with all the trimmings was served, followed by various local entertainment features, and concluded with the annual

dance. Dr. Dykstra presided as toastmaster. Among the distinguished guests whom he introduced, were Senator R. C. Regan, of Platte, co-introducer of Senate File 121, the so-called veterinary lien law enacted during the 1935 session of the Legislature, and Dr. J. C. Flynn, who gave a very able address, concluding by asking for the whole-hearted support of the entire veterinary profession toward supporting the A. V. M. A., the greatest organization of veterinarians in the world.

The election of officers for 1936 resulted as follows: President, Dr. G. H. Leenert, of Humphrey; vice-president, Dr. D. W. Hurst, of Tecumseh; and secretary-treasurer, Dr. J. D. Sprague, of David City.

E. C. JONES, *Secretary.*

Central Ohio Veterinary Medical Association

At the annual meeting of the Central Ohio Veterinary Medical Association was held at Otto's Inn, Watertown, October 4, 1935. 1935, officers for the year were elected as follows: President, Dr. R. J. Richards, Delaware; secretary, Dr. Harry A. Hoopes (reëlected), LaRue. Dr. Hoopes gave an account of the A. V. M. A. meeting in Oklahoma City.

Southeastern Wisconsin Veterinary Association

The annual meeting of the Southeastern Wisconsin Veterinary Association was held at Otto's Inn· Watertown, October 4, 1935. Officers elected for the year are: President, Dr. W. W. Arzberger, Watertown; vice-president, Dr. E. L. Morgenroth, Kewaskum; secretary, Dr. J. O. McCoy, Reeseville; treasurer, Dr. G. A. Gettelman, Hartford.

Miami Valley Veterinary Medical Association

Officers of the Miami Valley Veterinary Medical Association were elected at the quarterly meeting held at the Hotel Bancroft, Springfield, Ohio, October 23, 1935, as follows: President, Dr. Fred H. Dettman, Troy· Ohio; vice-president, Dr. T. W. Johnson, Dayton, Ohio; secretary-treasurer, Dr. M. C. Moses, New Carlisle, Ohio.

Dr. F. A. Zimmer, State Veterinarian, was the principal speaker. A turkey dinner followed the afternoon business session.

NECROLOGY

CHARLES ARTHUR WAGNER

Dr. Charles A. Wagner, of Williamsport, Pa., died very suddenly October, 3, 1934, according to a note recently received from his sister.

Born March 10, 1891, at Hazelton, Pa., Dr. Wagner attended local grade, high and business schools, before entering the University of Pennsylvania for the study of veterinary medicine. Following his graduation in 1914, he practiced for a short time. On December 1, 1916, he entered the service of the U. S. Bureau of Animal Industry, and was assigned to meat inspection at Chicago. On November 7, 1917, he resigned to enter the Veterinary Corps of the U. S. Army. He was assigned to duty with the 8th Cavalry at Marfa, Texas. On August 22, 1918, Dr. Wagner was reinstated in the Bureau of Animal Industry and assigned to meat inspection at Boston, Mass. He remained in the service until May 21, 1927. Some time later he entered the employ of the Pennsylvania Bureau of Animal Industry and served as senior field veterinarian for three years.

Dr. Wagner joined the A. V. M. A. in 1929.

WILLIAM F. TAYLOR

Dr. Will F. Taylor, of Athens, Mich., died in Aberdeen, S. Dak., April 27, 1935.

Born in Wakeshma Township, Kalamazoo County, Michigan, September 3, 1879, Dr. Taylor attended Athens local and high schools and then took a short preparatory course at Ypsilanti State Normal College. He taught for a time, but when his father died in 1902 he returned to the home farm, which he worked until 1916. He then decided to study veterinary medicine and entered the Grand Rapids Veterinary College. Following his graduation in 1918, Dr. Taylor entered the service of the U. S. Bureau of Animal Industry and was assigned to meat inspection at Chicago. He remained in the service until June

3, 1920. He reëntered the service at Sioux City, Iowa, September 18, 1934, and served until January 31, 1935.

Funeral services were held at Athens, Mich., with burial in the Factoryville Cemetery.

SYLVESTER JAMES MURRAY

Dr. S. J. Murray, of Le Sueur, Minn., died of a heart attack while making a professional call near Saint Thomas, Minn., December 17, 1935.

Born in Mapleton, Minn., July 24, 1870, Dr. Murray was graduated from the Ontario Veterinary College in 1894, and shortly thereafter located in Le Sueur. He was highly regarded as a citizen and held the position of municipal judge from 1923 up to the time of his death. All business houses in Le Sueur were closed during the funeral. Dr. Murray is survived by his widow (née Mary V. Rafferty), a son, a sister and two brothers.

J. P. F.

JOHN DOERR

Dr. John Doerr, of Melbourne, Iowa, died September 18, 1935. He had been in rather poor health for about six months.

Born in Grombach, Germany, August 16, 1879, Dr. Doerr attended school (gymnasium for six years) and then came to the United States (1902). He worked on a farm in Iowa for several years and then decided to study veterinary medicine at Iowa State College. He was graduated in 1911 and located at Melbourne for practice.

During the World War, Dr. Doerr was commissioned as second lieutenant in the Veterinary Corps, July 6, 1917; promoted to first lieutenant, September 11, 1918, and discharged on March 5, 1919. During this service he was assigned to duty at the Remount Depot, Fort Royal, Va.; Camp Dix, N. J., and Camp Dodge, Iowa. He held a commission as major in the Organized Reserve Corps.

Dr. Doerr joined the A. V. M. A. in 1924. He was a member of the Iowa Veterinary Medical Association. He is survived by his widow, two sisters and a brother.

EVERETT A. CHAPMAN

Dr. Everett A. Chapman, of Dodgeville, Wis., died September 15, 1935. Four days before his death he was taken ill, and two

days later was operated upon for appendicitis. He was a graduate of Iowa State College, class of 1934.

A. J. K.

CARL W. FISHER

Dr. Carl W. Fisher, who had been in general practice at San Mateo, Calif., for the past 34 years, died suddenly on November 26, 1935, while engaged in treating an animal at the Mills Estate, Millbrae.

Born in Cabot, Vt., February 23, 1871, Dr. Fisher first finished a course in veterinary medicine at the Ontario Veterinary College in 1898. Then he entered the New York State Veterinary College at Cornell University and was graduated in 1901. He immediately went to California and located at San Mateo.

During his professional career, Dr. Fisher became noted for his gentleness in handling animals. He achieved success not only in the treatment of diseases, but more particularly in their prevention. His success in developing and maintaining herds free from infectious diseases was well recognized. The efficiency and sincerity of his work was held in high esteem by his many clients in San Mateo County as well as by his veterinary colleagues throughout California.

Dr. Fisher joined the A. V. M. A. in 1903. He was a member of the California State Veterinary Medical Association, the Twelfth International Veterinary Congress and the San Mateo Lodge of Masons. He is survived by his widow (née Elizabeth J. Moore).

C. M. H.

ELI L. BERTRAM

Dr. Eli L. Bertram of Mount Healthy, Ohio, died at his home November 17, 1935. He was 62 years old.

Following his graduation from the Ontario Veterinary College in 1897, Dr. Bertram returned to his home in Strausstown, Pa., where he engaged in practice for a couple of years. On February 23, 1899, he entered the service of the U. S. Bureau of Animal Industry. He was assigned to meat inspection in Chicago, under Dr. O. E. Dyson, and remained as a member of the Chicago force for three years. He then was transferred to Davenport, Iowa, and later to Cincinnati, Ohio, and National Stock Yards, Ill. On December 31, 1919, he resigned from the

service to enter the employ of E. Kahn Sons Co. of Cincinnati, a connection which he retained until his death.

Dr. Bertram joined the A. V. M. A. in 1912. He is survived by his widow (née Cora Hope), three daughters and one son.

WALTER C. REEDER

An untimely death has removed one of Pennsylvania's loyal sons, Dr. Walter C. Reeder, of Springfield, Pa., who died at the Delaware County Hospital, December 2, 1935.

For many years Dr. Reeder had been giving his services to the furthering of education in the field of sanitary science, particularly milk hygiene. Alumni of the North Carolina College of Agriculture and Mechanical Arts, as well as the University of Pennsylvania, will recall his benevolent personality and his profound interest in sanitary work.

Born at Rising Sun, Md., on March 25, 1886, he was graduated from Maryland State College and subsequently taught high school in Maryland before entering the University of Pennsylvania. After completing the course at the School of Veterinary Medicine in 1912, he located at Elkton, Md., for general practice. In 1925, Dr. Reeder entered the service of the Suppiee-Wills-Jones Company. He was stationed first at various points in Delaware and for the past five years at Philadelphia. It was during this latter period that he became intimately associated with the faculty and students at Pennsylvania, where he assisted in the instruction in milk hygiene.

Dr. Reeder joined the A. V. M. A. in 1918. He held a commission as captain in the Veterinary Reserve Corps. He is survived by his widow (née Caroline McCudlough), a daughter, a brother and a sister. His passing is noted by all who knew him, socially and professionally, with a poignant sense of loss.

A. H. C.

JAMES E. McPHERSON

Dr. James E. McPherson, of Okmulgee, Okla., died in a hospital in Henryetta, Okla., December 6, 1935, as a result of an attack of heart disease. He was stricken very suddenly several days earlier while making a professional call in the vicinity of Henryetta, and was moved to a hospital there, where he remained until the day of his death.

Born on a farm near Kirklin, Hamilton County, Ind., July 6, 1891, Dr. McPherson attended local grade and high schools.

He then entered the Indiana Veterinary College and, following his graduation in 1915, he practiced at Windfall, Ind., in partnership with Dr. H. D. Yundt (McK '08).

About 1923, Dr. McPherson removed to Oklahoma and for about five years practiced as an assistant to the late Dr. C. R. Walter, at Tulsa. In 1928 Dr. McPherson removed to Okmulgee and entered practice on his own account there. He conducted a hospital for small animals in conjunction with his practice.

Dr. McPherson joined the A. V. M. A. in 1931. He was a member of the Oklahoma Veterinary Medical Association and had served in several important appointive offices. He was a member of the 12th International Veterinary Congress, an active worker in the Methodist Episcopal Church South, and was affiliated with the American Legion. He is survived by his widow, (née Harriette Jeanette Martin) and three sons.

C. H. F.

HOWARD DANNER

Dr. Howard Danner, of Indianapolis, Ind., died at his home September 22, 1935, after a long illness and following a major surgical operation. For the past 15 years Dr. Danner had been the Indiana representative of the Jensen-Salsbery Laboratories.

Born at Indianapolis, September 8, 1884, Dr. Danner attended local grade and high schools before entering the Indiana Veterinary College, from which he was graduated in 1906.

Dr. Danner joined the A. V. M. A. in 1927. He was also a member of the Indiana Veterinary Medical Association. He is survived by his widow (née Maymie Smelser), a sister and a nephew.

OTIS E. RAGSDALE

Dr. Otis E. Ragsdale of Lebanon, Ind., died at the Witham Hospital, December 13, 1935. Five days previously he had entered the hospital for an emergency operation for appendicitis. Peritonitis was the cause of death.

Born in Boone County, Ind., December 13, 1884, Dr. Ragsdale attended Thorntown grade and high schools and later Butler University. He then entered the Indiana Veterinary College and was graduated in 1912. He entered practice at Madison, Ind., later locating in Whitestown. Seventeen years ago, he removed to Lebanon. He established the Lebanon Electric Hatchery and operated it for several years. Dr. Ragsdale was considered quite an authority on poultry diseases.

Dr. Ragsdale was a member of Boone Lodge No. 9, A. F. & A. M.; the Masonic Chapter and Council, and Lebanon Commandery No. 43, of which he was a past commander. He is survived by his widow (née Charm Tucker), two daughters, one son, his mother, two brothers and four sisters.

A. F. N.

JOHN DAVID STILLWELL

Dr. John D. Stillwell, of Indianapolis, Ind., died at his home, December 21, 1935, after an illness of about six months.

Born May 18, 1875, at Ames, Iowa, Dr. Stillwell attended local schools and then entered Iowa State College for the study of veterinary medicine. He was graduated in 1905 and shortly thereafter entered the service of the U. S. Bureau of Animal Industry. Following the passage of the Virus-Serum-Toxin Act, March 4, 1913, Dr. Stillwell was placed in charge of the Indianapolis Virus-Serum-Control Station, which supervised plants licensed to produce veterinary biological products in Indiana, Illinois, Wisconsin, Michigan and Ohio. Dr. Stillwell continued in this work until recently, when he was retired from science for physical disability.

Dr. Stillwell joined the A. V. M. A. in 1917. He was a member of the National Association of B. A. I. Veterinarians and the Twelfth International Veterinary Congress. He is survived by his widow (née Nell Douglas Cole), three sons, three brothers and a sister.

Funeral services were held in Indianapolis and burial was at Ames, Iowa.

Mrs. Joseph P. Scott

Our sympathy goes out to Dr. Joseph P. Scott, of Kansas State College, Manhattan, Kan., in the death of his wife, Mrs. Lois Mary Scott, December 1, 1935. Besides her husband, Mrs. Scott left a daughter, Mary, and a son, Lawrence. At the time of her death, Mrs. Scott was enrolled as a sophomore in home economics at Kansas State College.

Sympathy is extended also to Dr. Gordon C. Kendall, in the death of his wife at Mayaguez, Puerto Rico, September 1, 1935; to Dr. Edward Lapple, of Saint Paul, Minn., in the death of his mother at Cincinnati, Ohio, December 2, 1935; and to Dr. and Mrs. P. O. Woods, of Greenville, Ill., in the death of their daughter, Mildred, at Hattiesburg, Miss., December 14, 1935.

PERSONALS

Dr. Joseph F. Knappenberger (K. S. C. '35) is now associated with Drs. Planz and Case, of Akron, Ohio.

Dr. Philip Forsberg (Chi. '14), of Hobart, Ind., has been reappointed Lake County Veterinarian by the Board of County Commissioners.

Dr. J. L. Bone (Chi. '91), of Bethany, Ill., was trampled by a cow he was treating recently and received a number of painful bruises.

Dr. Harry I. Goldwasser (Corn. '35), of New York, N. Y., is taking postgraduate work in pathology and parasitology at Ohio State University.

Dr. R. E. Nichols (Corn. '28), of Chatham, N. Y., is taking postgraduate work at Ohio State University. He is conducting research work on blood.

Dr. T. P. Gallahue (Chi. '17), of Waukegan, Ill., was reappointed Lake County Veterinarian by the County Board of Supervisors at the December meeting.

Dr. A. J. Striegel, (Chi. '18) has resumed his practice at Murphysboro, Ill., after spending some time in the employ of the U. S. Bureau of Animal Industry.

Dr. Robert Learmonth (Mich. '25) has returned to the University of Pennsylvania School of Veterinary Medicine, to resume his studies on bovine mastitis.

Dr. Geo. A. Ruggles (Colo. '33) has purchased the Western Veterinary Hospital at 8821 Aurora Ave., Seattle, Wash., from Dr. E. A. Schmoker (K. S. C. '17).

Dr. W. B. Van Cleave (Ind. '09), of Peoria, Ill., has been reappointed Peoria County Veterinarian, at a salary of $4,000 per year, by the County Board of Supervisors.

Dr. J. L. Ward (O. S. U. '35), after serving a temporary appointment in the U. S. Bureau of Animal Industry, has opened a hospital for pet animals at Lancaster, Ohio.

Dr. Fred W. Cruickshanks (Corn. '18), of Hagerstown, Md., has been critically ill with pneumonia, but was making slow progress toward recovery at the most recent report.

Dr. O. W. Nelson (Chi. '15), of DeKalb, Ill., addressed the local Rotary Club on December 12. His subject was "Animal Husbandry." Dr. Nelson is a past president of the club.

Dr. H. T. Clarno (Chi. '18) has resigned from the U. S. Bureau of Animal Industry after spending a year on Bang's disease eradication, and has reëntered private practice at Farmer City, Ill.

Dr. A. S. Houchin (U. P. '09), who has been practicing for a number of years at Strikersville, Pa., has returned to his former location, Newark, Del., where he is engaged in general practice.

Dr. Frank P. Mathews (Colo. '23), who has been in charge of poisonous plant investigations at the Texas Agricultural Experiment Station, Alpine, Texas, is spending a year at the University of Michigan, taking graduate work for a Ph. D.

Dr. William R. Teeter (Mich. '34) accepted an appointment in the U. S. Bureau of Animal Industry, on December 1, and is stationed at the Maryland Live Stock Sanitary Service Laboratory, at College Park, engaged in research work on Bang's disease.

Dr. G. A. Mills (Chi. '99), of Wausau, Wis., was elected president of the Board of Directors of the Marathon County Agricultural Society, at the annual meeting in November. Dr. Mills has been identified with the activities of the society for the past 35 years.